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THE SHATTUCK LECTURE.*

THE ETIOLOGY OF THE DISEASES OF THE CIRCULATORY SYSTEM.

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I THINK you will all agree with me that any existing classification of the diseases of the circulatory system leaves much to be desired. Writing of the best-elucidated form, acute endocarditis, Osler says,[1] "A good working classification, either etiological, anatomical, or clinical, is not easy to make." Turn the pages of any work on circulatory disease, to the affections of the myocardium, the so-called functional diseases of the heart, the diseases of the arteries, and you will search in vain for any single guiding principle in the arrangement of the great mass of facts accumulated by pathologists and clinicians.

I am not here to essay the impossible, to construct a single classification which shall embrace all of the diverse disturbances which may affect the circulatory system, or the equally impossible task of holding your attention for an hour while I do it. Every classification is tentative and temporary, but it has one definite object which cannot be achieved without it, the study of all the facts from a single point of view. The body is, to the anatomist, a complex of structures built up of tissues and their constituent cells; to the physiologist, of mechanisms and organs accomplishing transformations of energy by physical and chemical means. In the study of disease, the anatomically-minded pathologist considers lesions fundamental, the physiologically-inclined, their effect on function. The clinician needs both points of view, and neglects either to his peril. Diagnosis has its historic roots in pathological anatomy, and if they are cut, the tree dies; but the fruits of diagnosis, which are for the healing of the sick man's symptoms, grow on the many branches of exact functional study.

The student of disease faces another problem which does not exist for the investigator of the healthy organism, the ultimate problem of causation. Whence come these diseases, and why? What are their primary, and what their accessory causes? How do they reach their baneful development? On the finding of the right answers to these questions depends the final attainment of the goal of our profession, the prevention of disease. By as much as the proverbial value of prevention exceeds that of cure, does the importance of study of the causes of disease surpass that of its manifestations, its results, or its treatment. Clinical classifications should, therefore, be etiological whenever possible, to emphasize above all things the point of attack upon disease.

Every recent study of vital statistics has indicated clearly that the remarkable lowering of the general death rate during the past forty years has depended upon control of the acute infectious diseases and the lessening of deaths among children and young adults. While this

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has been in part the indirect result of improved living conditions, none will dispute the rôle which modern bacteriology and the discovery of the causes and modes of transmission of many of the infectious diseases has played in the achievement. On the other hand, sanitarians are now alive to the fact that the death rate for the age periods above forty has actually increased. This is brought out clearly in a comparison of the vital statistics of New York City for the years 1879-1881 and 1909-1911, compiled by Guilfoy.[2] While the child under five has an increased expectancy of life of 10.6 years now, as compared with thirty years previously, the adult at forty has a diminished expectancy of 0.5 years, and the aged live, on the average, 3.3 years less. Dublin,[3] studying the entire registration area of the United States, has shown an increased mortality from the age of fifty-five up in 1911 as compared with 1900. He has further proved clearly, as has Bolduan[4] from New York City, that the increase is due in the main to diseases of the heart, arteries and kidneys, and to cancer.

*TABLE I.—DEATH-RATE PER 100,000 OF POPULATION FOR CERTAIN CAUSES OF DEATH.—MALE AND FEMALE COMBINED.—(REGISTRATION STATES AS CONSTITUTED IN 1900.)

Cause of Death.	1900	1910	Per Cent. Increase.
1. Cancer (all forms)	63.5	82.9	30.6
2. Diabetes	11.0	17.6	60.0
3. Cerebral hemorrhage and apoplexy	72.5	86.1	18.8
4. Organic diseases of the heart	116.0	161.6	39.3
5. Diseases of arteries	5.2	25.8	396.2
6. Cirrhosis of liver	12.6	14.4	14.3
7. Bright's disease	81.0	95.7	18.1
Total	361.8	484.1	33.8

That diseases of the arteries have increased 396 per cent. in ten years seems to me impossible on its face. Those ten years witnessed the general adoption of blood pressure instruments by the practitioners of the United States. They also witnessed one of those extraordinary changes in diagnostic nomenclature comparable only to the revolutions in feminine fashions, which has made arteriosclerosis one of the most popular causes of death, and a diagnosis which any newspaper reporter considers a complete and satisfying explanation. With all such deductions, however, the statistics brought forward by Bolduan[4] from American sources show a definitely increased incidence of diseases of the circulatory system. The significance of the figures would be much more serious had the population remained homogeneous during this period. The influx of immigrants from the South European countries, however, introduces so large a variable, that all the facts of racial, social and industrial predisposition to circulatory disease, and of the difference in the age-distribution of the population would have to be worked out with great care before determining the actual increase of these diseases for any single racial stock, such as the native American of Anglo-Saxon origin, the Scandinavian, the North German or the Irish, which were the predominant stocks thirty years ago.

However men may differ as to details of interpretation, there can be only agreement as to the vast importance of the prevention of diseases which are responsible for approximately twenty-two per cent. of all the deaths in the United States. Public health officers[4] and insurance companies[5] are alike crying for the inauguration of a campaign against these diseases; but no campaign is justified because of the need for attaining an object unless the means for attaining that object are known and are available. Do we possess sufficient exact knowledge of the ultimate and the contributory causes of any of the diseases of the circulatory system to warrant widespread efforts to reduce their prevalence, or public propaganda for the dissemination of such useful knowledge? It is the attempt to answer these questions which has impelled me to bring before you this evening a sketch of the diseases of the circulatory system viewed from the standpoint of their etiology.

1. KNOWN BACTERIAL INFECTIONS.

(a) *Bacterial or septic endocarditis.*

The nature of the etiological agents has been more completely demonstrated for bacterial endocarditis than for any other lesion of the circulatory system. Post-mortem and clinical studies, and especially the blood culture studies of Lenhartz[6] and his pupils, Libman and Celler,[7] Horder[8] and others, have put the classification of this type of circulatory disease permanently upon an etiological basis, and have made its diagnosis possible wherever bacteriological blood examination is available. The acuter types due to haemolytic streptococci, the staphylococci, the pneumococci, occasionally the gonococcus or rarer organisms, are important rather as a special group of the septic diseases than as diseases of the circulatory system. They are uniformly fatal, and do not lead to chronic valvular disease. Their prevention is merely a small part of the large problem of prevention of sepsis. From the standpoint of the circulatory system, the chronic form of bacterial endocarditis due to attenuated streptococci is assuming constantly greater prominence. Not only may patients with such an infection survive for several years and present the evidences of valvular disease combined with mild symptoms of general infection, but the observations of Harbitz, Libman and others[9] have shown the possibility of healing of the lesions with disappearance of the bacteria. Infection by the attenuated coccus, now most commonly known as streptococcus viridans, has thus become one of the possible causes of chronic valvular disease of the heart. Improved methods of blood culture, such as have

[3] Dublin, L., Am. Jour. Pub. Health (n. s.), iii, 1264.

been devised by Rosenow,[10] have recently, in the hands of some workers, led to reports of the frequent detection of these organisms in cases of apparent benign endocarditis ending in recovery, with a permanent valvular defect. Oille, Graham and Detweiler[11] report so many positive findings that they have propounded the view that similar organisms are responsible for the common form of benign endocarditis. This I shall recur to later. So far as the general experience of clinical bacteriologists may be relied upon, proved bacterial endocarditis is one of the rare causes of chronic valvular disease.

Problems awaiting solution in this field concern the portal of entry for these bacteria and the conditions predisposing to their lodgment on or in the heart valves. These attenuated streptococci are present in the majority of normal mouths, are frequent inhabitants of the tonsil, of the intestinal tract, of abscesses about the roots of the teeth. Their discovery in these situations makes the theory that here is their portal of entry altogether plausible. Practice has not lagged behind theory, and physicians, laryngologists and dentists are now doing their best to eradicate them at all such possible portals. It is as yet too soon to form any estimate of the value of such measures. Certainly, once the infection of a heart valve has occurred, removal of the source of the infection is likely to be of little benefit. I have had a number of disappointing results from the attempted prophylactic removal of infected tonsils. I feel that it would be well, for a number of years, to have infected tonsils removed; that abscesses about the teeth should be evacuated for their own sake; that other known foci of infection should be cleaned up; but that one should preserve an open mind and wait, perhaps ten years, for the demonstration of the efficacy of these measures in the prophylaxis of bacterial endocarditis. The problem of the conditions predisposing to infection of a valve is of great interest, and the subject of active investigation at the moment, but no knowledge upon which action can be based is at hand.

(*b*) *Other bacterial infections of the circulatory system.*

Although we possess so much more detailed information about infections of the heart-valves that endocarditis seems incomparably the most frequent lesion of the circulatory system produced by the lodgment of bacteria, the other structures of the heart and the vessels may likewise suffer. In certain infections, lobar pneumonia especially, acute pericarditis occurs more frequently than endocarditis. As a cause of eventual cardiac disease, however, pericarditis does not seem to play a particularly important rôle.

Acute inflammatory changes in the myocardium, and even abscesses of the heart-wall, are observed at the autopsy-table in cases of sepsis. It must be exceptional that these are recovered from with the production of chronic lesions.

In bacterial endocarditis infected emboli may give rise to lesions of distant arteries, which develop into aneurysms. Very commonly, in the case of the streptococcus viridans, small emboli produce a now well-recognized lesion of the glomerular tuft in the kidney. This type of focal nephritis, however, does not appear to be associated with hypertension and secondary cardiovascular changes. The acute inflammations of the arteries have been the subject of much study since the original publication of Virchow[12] in the first volume of his Archives. Your own Dr. Mallory[13] has been a life-long advocate of the view that this is one of the important causes of chronic arterial disease. As with myocardial infection, so here the significance may best be discussed in the next section.

2. Probable, But Unproved, Bacterial Infections.

(*a*) *The rheumatic infections.*

No fact in clinical medicine is better substantiated than the association of benign inflammations of the endocardium, pericardium and myocardium with that infection which we must still be content to call rheumatic. While to Bouillaud belongs the credit of the first thorough description of the rheumatic lesions of the heart and the introduction of the term endocarditis, in his book published in 1835, those of us who cherish Anglo-Saxon tradition may remember with pride that Pitcairn, in 1788, taught his students about rheumatism of the heart in the wards of St. Bartholomew's Hospital; Matthew Baillie mentioned it in 1797 in the second edition of his famous "Morbid Anatomy"; and William Charles Wells,[14] in 1812, published an excellent series of case reports, including five with autopsies. For some unknown reason, possibly the failure carefully to observe the valves, pericarditis seems to have been disproportionately frequent during the earlier days of clinical and post-mortem study of the disease. At present it seems a very minor cause of circulatory death, the report of the Registrar-General for 1900[15] showing during the previous decennium 672 deaths from valvular disease in England, as against thirty-nine from pericarditis, calculated for one million living. The clear recognition of rheumatic myocarditis is of much more recent date, and due especially to the work of Krehl[16] and Romberg.[17]

Despite the long years of study of acute rheumatic fever and the many who have felt convinced that they have discovered its causative organism, no one is entitled as yet to speak with positiveness as to its etiology. I confess to entire openmindedness toward the subject. I do not believe that the clinical similarity of many cases of acute arthritis due to attenuated streptococci proves that these organisms are therefore the cause of the vastly larger number of cases of acute rheumatic polyarthritis in which they cannot be demonstrated. All of the recent experi-

mental work has been undertaken with a view to establishing the causative relation of organisms of the streptococcus-pneumococcus group with rheumatic fever. The recent work of Faber,[18] following up the earlier work of Loeffler, Cole and others, and his production of arthritis in sensitized joints, demonstrates how similar lesions may be produced with remarkable uniformity. It would also seem to throw light on the well-known tendency of acute arthritis to relapse; nevertheless, it still remains possible that true acute rheumatic fever is produced by an entirely different infectious agent.

In addition to the problem of its true cause, there are other problems capable of solution, which may clear the way for a prophylaxis of rheumatic heart lesions. These concern the portal of entry, the conditions predisposing to infection in general and to its localization in the heart in particular. The weight of clinical evidence certainly tends strongly to incriminate the throat as the portal of entry. Attacks are very commonly the sequel, after one or two weeks, of a tonsillitis or infection of the pharyngeal mucous membrane. Tonsillectomy is already much in vogue as a prophylactic measure. My opinion as to its usefulness here is the same that I have already expressed with reference to known streptococcus infection. My experience has taught me that it is not a prophylactic panacea; I even have grave doubts as to its usefulness after the individual has suffered from an attack of rheumatic fever, but I do not allow these doubts to prevent the patient from receiving what benefit the operation may give. Only a large statistical study covering a number of years can provide a basis of solid fact for or against the removal of tonsils.

General conditions favoring infection are too well known to require more than passing comment. The most striking is youth. I doubt whether any of us would recommend its prophylactic removal if we could. The colder climates seem clearly to predispose, and the late winter and early spring months. These influences are most interestingly discussed by Sticker[19] in his recent volume on diseases due to cold. No radical reduction in the amount of rheumatic infection seems likely to be achieved from anything short of the discovery of the portals of entry and how to close them, but I believe that children of families with known tendency to rheumatic infections may here and there be saved by us as individuals, if we give sufficient attention to these minor accessory causes. No theories concerning the cause of localization in the heart-valves have as yet led to practical results; neither the older ones, which laid stress on the mechanical factor, nor Rosenow's[20] ideas of organ specificity in the coccus.

(*b*) Benign endocarditis leading to chronic valvular disease is known to occur with considerable frequency in chorea, more rarely as a sequel of scarlet fever and throat infections. Other clinical associations are too rare to be of practical significance. Definite knowledge as to the etiological organism is still scantier here than for rheumatic endocarditis, though a streptococcus is occasionally demonstrated, and is most open to suspicion of guilt.

(*c*) Acute infectious myocarditis is a term very loosely used. Whether in most instances definite bacterial infection of the heart muscle can be assumed is very questionable. Degenerative changes in the muscle probably occur in most severe infections, actual inflammatory lesions in some. Diphtheritic myocarditis, one of the most important clinical types, seems clearly a toxic degeneration with secondary inflammatory reaction. During typhoid fever and pneumonia, the various exanthemata, and sepsis, and following influenza, the evidence of myocardial disturbance may develop and demand serious attention. To what extent such acute infectious myocarditis is responsible for chronic myocardial disease later in life, is a much-debated question. That some of these patients show progressive heart-muscle weakness must be granted; that any clear connection can be traced between the bulk of our cases of primary myocardial insufficiency and a preceding acute myocarditis, must be denied. For even the best authenticated type, rheumatic myocarditis, opinions differ sharply as to the effect of the lesion upon the functional integrity of the heart-muscle. Krehl[16] and Romberg[17] attribute to it the eventual failure of compensation in the affected hearts, while Aschoff and Tawara[21] hold that the scars left by the small focal lesions represent the destruction of so small a fraction of the heart-wall as to be without practical significance. Modern knowledge of such important specialized portions of the myocardium as the conducting system make it highly probable that even small scars may, in certain situations, lead to serious disturbances of rhythm. An excellent discussion of all the problems of acute and chronic myocarditis will be found in Krehl's classical work.[22] While emphasizing the lack of finality of our knowledge, I would not be thought to minimize the fundamental importance of myocardial lesions.

(*d*) Acute arteritis and phlebitis present a still more difficult etiological problem, of which the limits of this paper do not permit a complete discussion. The former may easily be admitted as a cause of more or less localized chronic arterial lesions, embraced under the general category of arteriosclerosis. Nothing is more difficult to trace than the ultimate responsibility for this disease of late life, when the accidents, strains and infections of half a century, in addition to the inherited quality of the arterial tissues, elastic and muscular, all afford plausible and almost unverifiable explanations of its origin. The problem is scarcely possible of approach from the clinical side, because the diagnosis of scle-

rosis of the aorta and visceral arteries is highly fallible, and even the discrimination of true arteriosclerosis of the accessible peripheral vessels difficult. At the present moment it is the fashion to detect thickening of the radial artery in nearly every adult male, and to index this as arteriosclerosis in hospital records. It is also the rule to consider hypertension and arteriosclerosis as synonymous, a tendency I wish, with Allbutt,[23] to combat vigorously. One of the most careful clinical studies, that made by Thayer[24] of the vascular complications and sequels of typhoid fever, showed that the radial arteries were far more frequently thickened, and the average systolic blood-pressure was higher in every decade, in patients with a history of previous typhoid fever. I have tabulated the histories of my series of hypertensive patients previously reported,[25] with reference to the incidence of past infections.

TABLE II.—HISTORY OF PAST INFECTIONS IN CASES OF HYPERTENSIVE CARDIO-VASCULAR DISEASE.

Infection.	Total Number Investigated.	Postive History.	
		No.	Per Cent.
Pyogenic infections, surgical, etc.	312	71	18.4
Typhoid fever	384	57	14.8
Pneumonia	379	49	12.9
Arthritis	374	46	12.3
Scarlet fever	377	37	9.8

The outstanding fact in this group is the frequency of typhoid fever compared with pneumonia. The mortality statistics for the whole registration area of the United States in 1913[26] give a death rate from typhoid of 17.9, and from pneumonia of 132.4. If one assumes a mortality of ten per cent. for the former and of thirty-three per cent. for the latter,—figures which certainly do not exaggerate the typhoid deaths,—the proportion of total cases would be, roughly, one of typhoid to three of pneumonia, a conservative figure. Yet more of my patients had passed through typhoid fever than through pneumonia. The relation is worthy of further critical study.

Faber[27] has studied this and all related questions exhaustively from the pathological side, and I would refer to his book for the collected facts and bibliography in this field, without entering upon further discussion of the effects of tuberculosis and other diseases. His conclusion is, that the greater the number of illnesses an individual has survived, the more arteriosclerosis he is likely to develop.

Periarteritis nodosa is a very rare acute or subacute inflammatory disease of the arteries. Its infectious agent is wholly unknown, but it is certainly not syphilitic, as frequently alleged.[28]

3. SYPHILIS.

(*a*) *Of the aorta.*

Proof of the syphilitic nature of certain frequent and important types of cardio-vascular disease is a recent achievement. Suspicion has long existed. Morgagni recognized the clinical association of aneurysm with syphilis, and Corrigan that of aortic insufficiency. Nevertheless, in spite of such occasional descriptions of syphilis-like lesions in the aorta as Heiberg's and Laveran's, and the important contribution by F. H. Welch[29] in 1875, showing the major rôle of syphilis in producing the aortic lesion leading to aneurysm, it remained for Doehle,[30] working under Heller in Kiel, to bring forward in 1885 a convincing histological study of the syphilitic lesion of the aorta. This was followed by a number of publications by Heller and his assistants. Even then it was not until a discussion before the German Pathological Society in 1903, by Chiari, Benda and Marchand, that the specific nature of the changes called syphilitic mesaortitis was generally accepted, and this diagnosis began to appear regularly in autopsy protocols. The further proof was afforded by Reuter[31] in 1906, who was able to identify the spirochetes in the aortic lesion. Important confirmation of this was brought forward by your own Drs. Wright and Richardson[32] in 1909. The indirect evidence afforded by the Wassermann reaction has accumulated rapidly since the first study of Citron.[33] The clinical diagnosis of syphilis of the aorta, as you all know, has now attained a degree of certainty at least as great as that of any other of the diseases of the heart and vessels. For admirable descriptions of the pathological and clinical aspects of aortic syphilis, and bibliography, I would refer to Gruber's monograph,[34] Fordyce's Harvey Lecture,[35] the excellent papers of Longcope,[36, 37] and Sir Clifford Allbutt's *magnum opus*,[23] among a host of others.

It is sufficient here to remind you of certain clinically important facts. Syphilis is a disease of the thoracic aorta, almost invariably stopping above the origin of the coeliac axis, and only rarely extending beyond the arch. The lesions are most marked at the root of the aorta, and, in a large proportion of the cases, involve the aortic cusps, producing an aortic insufficiency. Longcope[36] estimates that seventy-five per cent. of all aortic insufficiency in adults is due to syphilis. Aneurysm is the most serious result of the weakening of the aortic wall. This occurred in 24 of 120 cases of syphilis of the aorta studied post-mortem by Gruber,[34] in 48 of 248 patients observed by Stadler,[38] and in thirty per cent. of Symmers and Wallace's[39] seventy autopsies. Some dilatation of the aorta can be detected in practically every case examined roentgenologically. The relative frequency of syphilis of the aorta and other syphilitic lesions in the Johns Hopkins Hospital may be found in Tables III and IV.

(*b*) *Of the heart.*

In addition to syphilis of the aorta, actual syphilitic lesions of the heart-wall are clearly recognized. True gummata admit of no dis-

pute, but are manifestly rare. My impression, gained from reported cases and from my personal experience, is that their chief clinical importance lies in their frequent association with heart-block. As a cause of any other type of circulatory disturbance, gumma of the heart is insignificant. The relation of syphilitic infection to other myocardial lesions is a matter about which, at present, it is most difficult to speak with positiveness. None of these diffuse lesions is histologically specific for syphilis. There seems little question that a chronic fibroid myocarditis may result from the healing of gummata of the heart-wall, or, as a more diffuse process, may occur through the lodgment of the treponema pallidum without true gumma formation. Warthin[40] has described the finding of the organism in a large series of hearts of individuals dying not only with congenital, but also with acquired, syphilis. Associated with them were many different parenchymatous and interstitial lesions, and at times no obvious lesion whatever. Krehl[22] is of the opinion that syphilis is an important factor in the production of chronic myocardial disease, but actual demonstration is certainly lacking. The differential diagnosis from other types of myocardial damage is rarely possible, and I am quite convinced that it is not justified merely by the finding of a positive Wassermann reaction in a patient with myocardial insufficiency.

Narrowing of the mouths of the coronary arteries by syphilitic lesions of the root of the aorta is a not infrequent cause both of myocardial changes and of eventual cardiac death. Angina pectoris, whether we believe it dependent upon coronary narrowing, or, with Allbutt,[23] as due to the actual inflammation of the aorta, has a high incidence as a symptom in aortic syphilis.

Syphilis of the endocardium and pericardium is negligible.

(c) *Of the smaller arteries.*

The recognition of syphilitic lesions in the smaller arteries began with Virchow's first paper, already alluded to.[12] The history of the development of opinion in this field will be found in Allbutt's book,[23] presented by one who has made many contributions to its growth, and who was one of its earliest students. Syphilitic disease of the cerebral arteries, first thoroughly described by Heubner,[41] is, from the clinical standpoint, the most clearly recognizable type. It leads to such obvious and serious effects within a comparatively small number of years after the primary infection that the sequence is obvious. The majority of attacks of hemiplegia in young individuals are certainly syphilitic in origin.

About disease of the smaller arteries elsewhere in the body it is much more difficult to form an opinion. Current teaching, surely well represented by Osler,[42] makes syphilis "one of the most important single causes." This, however, is usually due to the inclusion of syphilis of the aorta in the general category of arteriosclerosis, a practice which should be abandoned. In a recent article Riesman[43] says, "Taking arteriosclerosis cases by and large, I may say that syphilis is the most frequent cause of the uncomplicated type of the disease." But arteriosclerosis almost inevitably involves many complications, and he is certainly confusing arteriosclerosis with essential hypertension. Mott,[44] in a compendious discussion, speaks of syphilis as affecting the arteries generally, but gives specific facts only for the aorta, coronary and cerebral arteries, and in rare cases the pulmonary. Herxheimer's authoritative review[45] shows the occasional report of true syphilis of the larger peripheral arteries, and of widespread arterial lesions, but these belong to the exceptions. No facts have yet been brought forward to prove that syphilis is of more importance than other chronic infectious diseases in the etiology of ordinary arteriosclerosis.

Recently Stoll[46] has published interesting observations on the rôle of syphilis in hypertensive cardio-vascular disease. He has made the suggestion, of great importance if it can be confirmed, that the well-known cardio-vascular heredity is "familial cardio-vascular syphilis." He has shown how much more frequently a syphilitic taint, affecting several members of a family, may be detected, if the possibility be in mind, and both the patient and his family be investigated with intelligence. On the other hand, much of his argument rests upon the validity of Noguchi's luetin test as evidence of otherwise latent syphilis. This has already been seriously questioned by Sherrick,[47] who showed the remarkable effect of potassium iodide in producing a positive reaction. Sherrick's results have been confirmed in a series of cases this winter by Rivers in our clinic, which will appear later. He has demonstrated to our satisfaction that the test has no diagnostic value in any patient that is receiving iodide.

The whole matter has seemed to me of such significance that I have made it the subject of a special study of the medical patients of the Johns Hopkins Hospital between September 21, 1914, and April 2, 1916. For assistance in this I have to thank the members of my staff in general, and Dr. Mary A. Hodge and my secretary, Miss Lauer, in particular.

While the chief significance of these figures is clear, certain comments are necessary. The relation of white and colored populations in Baltimore, as given by the United States Census of 1910, was 84.8% white to 15.2% colored. This is practically identical with the proportion of white and colored patients in the hospital, and the morbidity rates of the hospital should, therefore, be comparable with the registered deaths of the city as a whole. On the other hand, it must be remembered that the white patients of the hospital, including, as they do, a number of

TABLE III.—INCIDENCE OF CERTAIN DISEASES OF THE CIRCULATORY SYSTEM AND OF WASSERMANN REACTIONS IN PATIENTS OF THE MEDICAL CLINIC OF THE JOHNS HOPKINS HOSPITAL, SEPT. 21, 1914, TO APR. 2, 1916.

	White.		Colored.		
	No.	Per Cent.	No.	Per Cent.	Total.
1. Admissions	2029	85.4	347	14.6	2376
2. Individuals	1884	85.1	329	14.9	2213
3. Patients having Wassermann reaction done	1272	....	288	....	1560
4. Patients with W. R. positive	166	....	124	....	290
Per cent. of 2	....	8.8	...	37.6	....
Per cent. of 3	....	13.	...	43.	....
5. Syphilis of aorta:					
Per cent. of 2	31	1.6	50	17.4	81
W. R. positive	25	....	47	....	72
W. R. negative (aneurysms*)	6	19.3	3	6.	9
6. Other aortic lesions†					
Per cent. of 2	21	1.1	6	1.8	27
Dilated aortic arch	14	....	4	....	18
Aortic insufficiency	7	....	2	....	9
7. Chronic endocarditis, all valves. Per cent. of 2	45	2.4	7	2.1	52
8. Hypertension, all cases:					
Per cent. of 2	250	13.7	70	21.2	320
W. R. done	230	....	69	....	299
W. R. positive	8	....	22	....	30
Per cent. of W. R. cases	....	3.5	...	31.9	....
Per cent. of all cases	....	3.2	...	31.4	....
9. Myocardial insufficiency, all cases:					
Per cent. of 2	168	8.9	82	24.9	250
W. R. positive	17	10.1	30	36.6	47

* Cases of thoracic aneurysm, unquestionably syphilis of aorta. This represents the minimum of negative reactions in syphilis of aorta. A study of the reaction in all cases going to autopsy since 1908, by Dr. Mary A. Hodge, which will appear subsequently, shows approximately 70 per cent. positive and 30 per cent. negative Wassermann reactions at the Johns Hopkins Hospital for the entire period, in cases of proved syphilis of the aorta.

† Includes arteriosclerotic lesions, chronic endocarditis, etc. Probably includes cases of syphilis of aorta not clinically recognizable as such, in which no autopsy proof was available.

TABLE IV.—ANALYSIS OF CASES WITH POSITIVE WASSERMANN REACTION IN TABLE III.

Diagnosis.	White.	Colored.	Total.
Syphilis:			
Of aorta only	21	28	49
Of aorta and central nervous system	3	6	9
Of aorta and central nervous system; hypertension	..	1	1
Of aorta; other visceral, bone or skin lesions	..	2	2
Of aorta; chronic nephritis; hypertension	..	3	3
Of aorta; hypertension	1	7	8
Total of aorta	25	47	72
Of central nervous system only	86	7	93
Of central nervous system and aorta	3	6	9
Of central nervous system and aorta; hypertension	..	1	1
Of central nervous system; other visceral, bone or skin lesions	2	2	4
Of central nervous system; chronic nephritis; hypertension	..	3	3
Of central nervous system; hypertension	5	1	6
Total of central nervous system	96	20	116
Of heart; heart-block	..	1	
Of carotid, aneurysm	..	1	2
			190
Deduct duplicates	3	7	10
			180
Other localization only; visceral, bone, skin, etc.	21	12	33
Total syphilitic lesions			213
No definite syphilitic lesions. Latent syphilis:			
Chronic nephritis; hypertension	1	5†	6†
Hypertension	1	1	2
Diabetes	3	..	3
Pneumonia	3	9	12
Other acute and chronic diseases	19	35	54*
Total	27	50	77
Total positive Wassermann reactions	166	124	290

* Including two cases of possible syphilis of the myocardium. † Including two cases of possible syphilis of the aorta.

TABLE V.—ANALYSIS OF 299 CASES OF HYPERTENSION IN TABLE III.

	White. No.	White. Per Cent.	Colored. No.	Colored. Per Cent.	Total.
1. Evident renal involvement:					
Wassermann negative	121	....	26	....	147
Wassermann positive	1	0.8	11	29.7	12
Total	122		37		159
With syphilis of aorta or nervous system	1	....	6	....	7
Without discoverable syphilitic lesions	...	0.0	*5	13.5	*5
2. No evident renal involvement:					
Wassermann negative	101	....	21	....	122
Wassermann positive	7	6.5	11	34.4	18
Total	108		32		140
With syphilis of aorta or nervous system	6	....	10	....	16
Without discoverable syphilitic lesions	1	0.9	1	3.1	2

* Of these cases, two had possible syphilis of aorta.

private patients, come from a wide area, and frequently for diagnosis, rather than for the treatment of serious illness. They represent, therefore, a partially selected material, and the graver disorders of the circulation would thus tend to higher proportional representation in the colored wards. Furthermore, the racial division represents an industrial one as well, the colored race being the manual laborers. The total deaths and death-rates of Baltimore for 1913[26] were:

White	7912	16.2 per 1000
Colored	2712	31 per 1000

To bring out the relative importance of circulatory as contrasted with infectious diseases as a cause of death in the two races, I have made the following table from the recorded deaths of the Mortality Statistics,[26] pages 252 and 253.

TABLE VI.—DEATHS IN BALTIMORE, 1913, FROM CERTAIN IMPORTANT DISEASES.

Deaths from:	White. No.	White. Per Cent. of all White.	Colored. No.	Colored. Per Cent. of all Colored.
Tuberculosis (lungs)	742	9.4	452	16.7
Pneumonia	780	9.8	393	14.5
Bright's disease	801	10.1	234	8.6
Organic heart	706	8.9	268	9.9
Cerebral hemorrhage, etc.	440	5.5	102	3.7
Cancer	510	6.4	91	3.3

For the city as a whole, therefore, a larger proportion of all white persons die of diseases of the heart, arteries and kidneys, than of colored persons. Our hospital figures must then be due to selection of material, the colored patients seeking the hospital only when unable to work. In addition, the number of negroes living at the higher age period of circulatory disease is probably less.

That the colored patients suffer from syphilis of the aorta ten times as frequently as the white, cannot be due wholly to selection. Should this fact, and the finding of a positive Wassermann reaction in approximately forty per cent. of the colored patients, be interpreted as demonstrating so extensive a syphilization of the colored race in our cities? I think not without deductions. Clearly my figures are interdependent. The proportion of patients with positive Wassermann reaction is more than a third due to the presence of the patients with aortic syphilis. The negro, being a manual laborer, may, and I believe does, have a greater liability to the development of aortic lesions, and especially of aneurysm, if he contracts syphilis. A recent article by Boas[48] discusses the prevalence of syphilis in the two races and faces the same difficulties of interpreting hospital statistics, finally concluding that syphilis is from one and a half to two and a half times more frequent in negroes, but that its frequency in them, sometimes estimated at 75%, has been grossly exaggerated.

From another point of view, the great preponderance of tuberculosis and pneumonia as causes of death in the negroes, and their low cancer rate, argue against the predominant influence of syphilis. Grotjahn[49] cites the experience of the Gotha Life Insurance Company with syphilitics as follows: The mortality from all causes was 68% above that of all insured. The deaths among them were higher by 10% from infectious diseases, 60% from malignant growths, 64% from kidney disease, 116% from cardio-vascular disease, 128% from apoplexy, and 145% from mental diseases, exclusive of paresis. A comparison of these figures with mine shows that racial susceptibility must play at least as important a rôle as syphilis in determining morbidity among the negroes of Baltimore.

When we turn to the particular question of the rôle of syphilis in hypertensive cardio-vascular disease in the white race, the very low percentage of positive Wassermann reactions, at the most 3.5%, compared with the figure for the whole number of white patients, 8.8%, seems to

give an effectual answer in the negative. This is in accord with the experience of Walker and Haller[50] at the Peter Bent Brigham Hospital.

The only argument possible is from the greater frequency of this type of circulatory disease in the colored wards. Even here the proportion of positive Wassermann reactions is less than the average for these wards. If we turn to Table V, where I have shown the combination of hypertension and syphilis of the aorta or central nervous system, there remain, after deducting such cases, only 13.5% of the cases with renal involvement, and 3.1% of those without, whose positive Wassermann reactions could not be explained by obvious syphilis elsewhere.

My private cases are unsatisfactory as evidence, since they were, for the most part, before the days of Wassermann reactions. Of the more than 400 patients, only 23 gave a history of syphilis. This merely indicates the difference between hypertension and such diseases as tabes, long known to be related to syphilis because of the frequency of a syphilitic history, and called para-syphilitic until modern investigation proved their true nature. Our predecessors never included cardio-renal disease among the para-syphilitic manifestations.

My conclusion is, that no evidence has been brought forward to place hypertensive cardio-vascular disease in the category of the direct results of syphilis, and that syphilis plays an indirect rôle, if any, in its causation.

4. Rare Infections.

Of the rarer infections of the circulatory system, tuberculosis alone deserves consideration. Tuberculous pericarditis may occur as a clinically primary disease. Other tuberculous lesions of the heart are of purely pathological interest.

5. Parasites and Tumors.

Parasites and tumors, involving the heart or large vessels, need be mentioned merely for the sake of completeness. They may delight the custodian of a pathological museum, but not the practitioner or the sanitarian.

6. Intoxications.

(*a*) *Exogenous.*

It is difficult to form any just estimate of the part played by poisons in the etiology of cardio-vascular disease. Unlike the nervous system, where the various metallic poisons each produce specific types of disease, due to selective action on different structures, no definite types of circulatory disease can be distinguished as due to particular intoxications. Perhaps the nearest approach to this is the influence of lead on the arteries. In acute lead poisoning, with the colic, there is regularly vascular spasm and hypertension. In chronic plumbism, I have occasionally seen the picture of hypertensive cardio-vascular disease, with arteriosclerosis, and I am personally convinced that lead is one of the rarer, but most definite, causes of it.

The rôle of alcohol in the production of disease of heart or vessels is a burning question of the day. There can be no doubt that the death-rate from circulatory disease is considerably higher in those occupations where habitual drinking is the rule, as the statistics of Sendtner[51] for Munich show. In 1889 the death-rate from heart diseases was 11% for all adult males, 21% for male, and 19% for female innkeepers, and 16% for brewery employees. On the other hand, Tatham,[52] in studying the death-rate of various classes employed in the manufacture and sale of liquor in England and Wales, found a similar greater incidence of circulatory death, but also an increased rate from diseases of other systems, from tuberculosis and influenza, from many other conditions, and from all causes combined. The deleterious effects of habitual drinking are obvious. The specific effect of alcohol, apart from other associated harmful influences, upon heart or vessels, I am not prepared to express any opinion about.

The most commonly assumed example is the so-called "Munich or Tübingen beer heart." But in this, excessive muscular work and the consumption of large quantities of fluid surely so complicate the interpretation as to leave the influence of the alcohol beyond computation.

Alcohol is frequently assumed to be one of the important causes of arteriosclerosis and its associated cardio-vascular disturbances. Allbutt[53] (page 246), reviewing the evidence, including the valuable negative findings of Richard Cabot,[53] concludes that alcohol is not "a cause in eminent domain," but acts as the ally of other poisons. Faber[27] believes it exerts a deleterious influence on the vessels directly as well as indirectly. A study of my own private cases showing hypertension lends no support to the view that alcohol has an important influence in the production of this type of arterial disease. Of 397 patients whose histories as to alcohol were known, 37.5% were total abstainers; 31.5% took alcohol only occasionally in small amount; 18.1% were habitual temperate drinkers, that is, never using more than three drinks a day; those who regularly or occasionally used alcohol in excessive amounts made but 12.9% of the total.

Coffee and tea are commonly used poisons, perhaps less commonly abused than formerly. That they may lead to disturbance of the heart's rhythm, to vaso-motor disorders and to marked subjective symptoms is well known. It is possible that permanent lesions in the heart muscle may follow, but evidence is very difficult to obtain.

Tobacco is equally known to produce marked disorders of cardiac rhythm, especially extra-systoles, and in some individuals smoking will raise the blood-pressure. The tobacco-heart is, in the popular mind, a well-defined etiological

type of circulatory disease. Krehl[22] believes in a toxic angina pectoris due to tobacco misuse. I have seen cases which impressed me as such. Allbutt[23] and Osler[24] think it rare. In order to obtain concrete facts on which to base an opinion, I have tabulated the histories as to tobacco of 226 men with anginoid pain, seen by my father or myself, and, as a comparison, of 285 men with hypertension, and of 300 other male patients. Certainly no important influence of tobacco can be seen in these figures.

Plummer[55] has brought forward interesting evidence of the association of hypertension with old goitres of his so-called non-hyperplastic type. Eighteen per cent. of such patients studied by him, who were more than forty years of age, showed a systolic blood pressure of above 160. Looked at from the other point of view, a survey of 423 of my hypertensive patients, with reference to this point, showed only two with goitre associated with some toxic symptoms, in all less than 2%. In the same

TABLE VII.—THE USE OF TOBACCO IN MEN WITH HYPERTENSION OR ANGINA PECTORIS, AND IN ALL OTHER MEN.

	Hypertension.		Angina Pectoris.		Other Patients.	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
No tobacco	59	20.1	49	21.7	80	26.7
Not over three cigars a day	101	36.	60	26.5	78	26.
Four to six cigars	76	26.7	63	27.9	69	23.
Over six cigars	49	17.2	54	23.9	73	24.3
Total	285	100.0	226	100.0	300	100.0

I have been struck with the frequency of temporary discomforting palpitation, with extrasystolic irregularities in physicians, following the excessive use of coffee and tobacco during periods of extreme overwork, the symptoms all disappearing with the resumption of temperate habits. I have seen auricular fibrillation supervene.

It is safe to say, therefore, that toxic cardiac and vaso-motor disturbances are well authenticated; that they are evidenced for the most part by disorders of rhythm, and are attended by striking subjective symptoms; that they may lead to chronic myocardial disease is a plausible hypothesis, which can neither be affirmed nor denied in the present state of our knowledge; that they may lead to chronic arterial disease is at present still more conjectural.

(*b*) *Endogenous.*

The most striking and best known example of circulatory disease due to an intoxication, arising from the perverted activity of an organ of internal secretion, is the cardiac disorder of exophthalmic goitre. In its milder forms or its earlier stages, characterized merely by an exaggerated cardiac activity with increased rate and a tendency to peripheral vaso-dilatation, it leads, in its graver forms, to severe, and even fatal, myocardial insufficiency, with extrasystolic arrhythmia or eventual fibrillation of the auricles. Thyroid intoxication is, therefore, a clear cause of a definite type of circulatory disease, and one may with justice speak of a thyreotoxic cardiopathy if one wishes to think in etiologically exact, though somewhat pedantic, categories. A somewhat similar, though less adequately differentiated type of cardiac disturbance, is seen in association with uterine myomata, the so-called "Myomherz" of the German writers.

group were 12 with myomata, out of a total of 151 women, nearly 8%.

The last twelve years have seen numerous attempts to connect permanent hypertension with over-function of the adrenal gland. The hypothesis is a most attractive one, the indirect evidence for it considerable. The extreme antithesis of hypertensive cardio-vascular disease is seen in the vascular asthenia and hypotension of Addison's disease or following the experimental removal of both adrenals. Hypertension is regularly absent in chronic nephritis only in the peculiar type associated with general amyloidosis, and in this the adrenals are extensively infiltrated with amyloid. The blood sugar curve in patients with chronic nephritis has this winter been shown by Hamman[56] in our clinic to be very similar to that of diabetes, and not easily interpreted as mere renal retention. On the other hand, since I reviewed this whole subject in 1913[57] there has not been a single additional piece of direct evidence in support of the hypothesis. It remains, therefore, an interesting etiological speculation, still deserving of further experimental investigation, and nothing more.

The cause of arteriosclerosis and of hypertension is frequently sought in poisons having their origin in a deranged body-chemistry. Clinical observation has long associated arteriosclerosis with diabetes as a definite complication, and responsible for the not uncommon myocardial insufficiency of elderly diabetics through implication of the coronary arteries. Hypertension is also fairly common in diabetic patients. Of my 458 carefully-studied private patients with permanent high blood-pressure, 44, or 9.6% were diabetics. Of 320 hypertensive patients in the Johns Hopkins Hospital since September, 1914, 23, or 7.2%, were diabetics. The figures are equally convincing when computed for the incidence of arterial disease in diabetes. Beyond

the mere fact of clinical association, however, it is not possible to go. Whether the arterial disease is produced by the hyperglycemia or other chemical derangements of diabetes, or is dependent upon some phase of the disorder of the internal secretions which lies back of the diabetes, or bears some other relation of causation or coincidence, must be left for the future to determine.

Gout, especially in England, has been widely assumed to play an important rôle in the etiology of the disease which, from the standpoint of the cardio-vascular system, is called hypertension, with its resultant hypertrophied heart and arteries, from the standpoint of the kidney, chronic interstitial nephritis. In fact, the primary type of the latter is, in England, called the gouty kidney. It must be borne in mind that gout is a very common disease in London, and a comparatively rare one in this country. Of my private patients with hypertension, 22, of 423 in whom this fact was noted, either themselves had gout, or had a distinct family history of it. The maximum incidence of gout in the etiology of the disease in these patients was, therefore, 5.2%. Of the 320 hospital patients, only two had definite, and one other possible, gout. During the same period two patients with long-standing gout have died of other diseases, showing functionally normal kidneys during life and no chronic interstitial nephritis at autopsy. I do not, therefore, believe that a gouty nephritis or a gouty arterial disease can be recognized as an etiological type, but that gout may be reckoned among the predisposing factors favoring the influence of the real, but unknown, causes of these diseases.

The association of chronic renal disease and permanent hypertension leading to hypertrophy of the heart, to thickening of the larger arteries, and the eventual development of arteriosclerosis, terminating frequently in myocardial insufficiency or the rupture or occlusion of a cerebral artery, has been a commonplace of clinical and pathological observation since its first description by Richard Bright. It is one of the best-differentiated clinical types of circulatory disease, and one of the commonest. How should it be classified etiologically? The discussion of this problem alone would require more than the space of a single lecture, and would lead, at best, merely to a re-statement of conflicting hypotheses. If you will allow me to be a bit dogmatic in order that I may be decently laconic, I would say that only in the minority of cases should the disease be thought of as primarily renal, and the circulatory disorder as secondary thereto. Such a case as that described by Mann[58] shows how a primary scarlatinal nephritis may, after decades, result in secondary contracted kidney with true nephritic hypertension and hypertrophied heart, but these are very rare. Of 379 private patients investigated for this point, only 16, or 4.2%, of those with hypertension had ever suffered from acute nephritis; and of the 320 hospital cases, but three gave such a history. Of these 320, 61 were assumed to have an inflammatory renal disease and indexed as chronic diffuse nephritis with hypertension. Among these were 21 women, four of whom had shown the symptoms of nephritis during preceding pregnancies, or 19%. From this figure and from experience in other cases coming to autopsy, I believe that pregnancy may safely be considered one of the causes of progressive renal disease leading to hypertension, whether through metabolic poisons or not, we lack any information. Of the true causes of most cases of chronic diffuse nephritis, I feel safe in asserting that we are almost wholly ignorant.

In the majority of patients with so-called cardiorenal disease, I am convinced that it is essentially a disease of the circulatory system, involving the kidney through its vascular apparatus, and sometimes sparing it entirely. The behavior of the blood-pressure in these patients, when seen early, strongly suggests that the hypertension is dependent upon an exaggerated vaso-constrictor irritability, and that the development of the lesion in the arterioles comes later. This is Allbutt's "hyperpiesia,"[23] Huchard's "préselerose."[59] Volhard and Fahr[60] speak of the whole disease as "benign essential hypertension." No name will be satisfactory until it rests upon an etiological basis. Allbutt's designation is merely hypertension in unfamiliar form. His assumption of true plethora, increased blood volume, has never been proved constant in these patients. Observations by Keith, Rowntree and Geraghty[61] in our clinic failed to show it in any of the cases examined by their accurate method. Allbutt looks upon the disease as distinctly one of overfeeding. Of 301 of my private patients in whom this could be ascertained, 126, or 41.8%, committed dietary excesses. The remainder were entirely temperate eaters. The figure, however, is not without significance. The frequency of the disease in our colored wards, however, shows that the overfed have no monopoly of it. Were I here to speculate, I would venture the opinion that a chronic intoxication will eventually be proved underlying this condition; but whether the poison originates in disturbed metabolism, in deranged or excessive function of a gland of internal secretion, or otherwise, I should not even hazard a guess.

7. Nutritional Disturbances.

In this category come the brown atrophy of the heart seen in so many wasting diseases, which is clinically but part and parcel of the underlying disease, and the much more important effects of anemia upon the heart muscle. These are too well known to need more than passing comment. Their importance lies in the clear recognition that anemia entails definite

myocardial weakness and that safeguarding of the heart is necessary. From time to time severe grades of myocardial insufficiency develop as a definite result of anemia. On the other hand, it is necessary to bear in mind that the cardiac murmurs which occur in such hearts indicate only the temporary weakening of the heart muscle, and not permanent damage to the valves.

It is quite likely that the myocardial insufficiency, affecting chiefly the right heart, which accompanies beri-beri and scurvy should be considered nutritional in origin. In addition, the minor evidences of muscular exhaustion, to use Mackenzie's phrase, seen in women after prolonged lactation and too frequent child-bearing, are probably of this nature.

Viewed from the standpoint of the heart alone, the local nutritional disturbance resulting from narrowing of the coronary arteries ranks high among the causes of fatal cardiac disease in the elderly. From the standpoint of general circulatory etiology, however, it belongs with the rest of arteriosclerosis, and not in the category of nutritional disorders.

Obesity is not without influence upon the cardio-vascular system. It is not uncommon in the anamnesis of patients with hypertension, 15.7% of my patients, and 29.3% of my hospital patients exhibiting it. On the other hand, it must be borne in mind that these patients come from the age-periods in which obesity is common. Mr. Frederick L. Hoffman, statistician of the Prudential Insurance Company, has kindly furnished me with estimated figures for the United States, showing that above forty, 22 to 24% of the males, and 26 to 28% of the females are 10% or more above the average in weight.

That the obese often suffer from myocardial insufficiency is well known. This clinical association should be thought of as the weak heart of the fat rather than as the fatty heart. Two elements probably enter into it,—an actual impairment of the heart muscle, and the effect of the permanent increase in cardiac load.

8. Mechanical Disorders.

In this category I would embrace all those circulatory disorders which have their origin in increased demands upon the circulation, that is, the structural alterations resulting from overwork. The clearest examples of this are seen in those patients who develop a hypertrophied right ventricle as a result of alterations in the pulmonary circulation produced by emphysema, chronic fibroid tuberculosis of the lungs or extreme kyphosis. Many of these patients die a cardiac death from the eventual failure of the hypertrophied right ventricle to meet the constantly growing demands. The analogous condition for the left heart is myocardial failure consecutive on permanent hypertension. But, as I have already said, the fundamental disease is usually circulatory, and its etiology must therefore be carried back one step farther, to the cause of the hypertension. From the standpoint of the heart, however, it is an overwork disease.

The question of so-called acute dilatation, over-strain of a sound heart from excessive muscular work, has been much debated, and must still be considered open to argument. Heart muscle and skeletal muscles may usually be assumed to develop in balanced proportion, so that the latter cannot demand of the former more than it is capable of, unless it be diseased. The otherwise unexplainable myocardial failure seen in men doing heavy manual labor, and usually drinking large quantities of fluid, may well be considered an overwork disease. I have seen a number of such patients with marked hypertrophy and dilatation of the heart at autopsy, who had only chronic passive congestion of the kidneys, and whose blood-pressure was normal during life. This is the so-called "beer heart" of the Germans, but beer is neither a necessary element in its causation nor an adequate term for its etiological classification. In the same category, I believe, the so-called polycythemia hypertonica of Geisböck[62] should be placed.

Can we affirm a similar pathological change in the arteries arising from overstrain? This has long been a vexed question. George Johnson[63] ascribed the thickened arteries so common in hypertensive patients to hypertrophy of the media. Allbutt[23] looks on the eventual development of arteriosclerosis in hypertension, his hyperpiesia, as the result of strain; and Adami[64] believes strongly in mechanical strain as a cause of fibrosis, and even the Mönckeberg arteriosclerosis. Thoma's[65] mechanical theories of arteriosclerosis are well known, and Jores[66] admits the probability that hypertension precedes the development of the elastic and intimal lesions in the arterioles. It is clear to me that Allbutt is right in his view that the diffusely thickened arteries of hypertensives are different from the beaded, calcified arteries of the aged, the former representing a compensatory reaction under stress, the latter a degenerative process. His designations for these two types of arteriosclerosis, hyperpietic and decrescent, represent a real attempt at etiological classification, though the names are not likely to become popular. Faber[27] unites both types in his view that arteriosclerosis arises from a disproportion between the strength of the vessel-wall and the blood-pressure. The mechanical factor is particularly clear in the cases of pulmonary arteriosclerosis associated with mitral stenosis. As a secondary factor, producing myocardial failure in a damaged heart or aneurysm in a weakened syphilitic aorta, mechanical strain plays a large part, often ushering in the final stage of circulatory disease. It probably determines the localization of arteriosclerotic changes in many in-

dividuals. Romberg[67] has said: "A man develops arteriosclerosis predominantly in the vascular areas which he has most taxed."

9. Nervous Disorders.

The list of nervous disturbances of the circulation, formerly long, has been wonderfully abridged by the newer physiology and its myogenic theory of the cardiac rhythm. There remain, as true nervous disturbances of the heart, only those more or less transient disorders of rate and rhythm due to influences reaching the heart through the extracardiac nerves, and the important part played by the higher nervous centers in determining the character of symptoms. Whether there are primary diseases of the vegetative nervous system with circulatory manifestations is not certain. Cannon and Fitz's[68] recent work suggests the possibility that, back of the overactive thyroid in exophthalmic goitre, may lie a still more fundamental disorder of the sympathetic.

As to tonus disturbance from nervous influences we are still more in the dark. States of vaso-motor instability, with local or general symptoms, are, of course, familiar; but only as clinical pictures without adequate explanation. Friedrich Müller[69] believes that nervous individuals are more prone to develop arteriosclerosis.

Cardiophobia, so frequent among medical students, is no circulatory disease, yet it should be borne in mind by all who plan propaganda for arousing the community to the dangers of circulatory disease. They must be prepared to treat it promptly and effectively.

10. Defects of Development.

The various errors of development of the heart and great vessels make a chapter in cardiovascular pathology of fascinating interest to the embryologist, but afford no material for a campaign of prevention, and may be dismissed as a numerically infrequent cause of cardiac death.

11. Hereditary Disease.

The belief in an inherited quality of the arterial tissues with a tendency to premature death from apoplexy, angina pectoris, or other local manifestation, is too firmly grounded in clinical observation to be without basis. Certain families show a striking preponderance of such deaths. Hypertensive arterial disease must be looked on today as the type in which heredity plays the largest rôle. Apart from such remarkable family histories as I could cite, however, my whole group of private patients gave the cause of death as cardio-vascular for both parents, or one parent and other members of the immediate family in 22%, for one parent or brother or sister in 28%, while 50% had no cardio-vascular heredity at all. Such figures suggest a subordinate and accessory influence of inheritance for the majority of patients.

12. Relative Importance of the Various Causes of Myocardial Insufficiency.

Finally, I wish to call attention to the relative importance of some of the causes we have examined. For this purpose I have tabulated all the cases having myocardial insufficiency observed in our medical wards during the period already mentioned.

Table VIII.—Analysis of the Causes of Myocardial Insufficiency and Their Distribution by Race.

Total Patients, 1884.	White.		Colored.		Total.	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent. of all Causes.
1. Hypertension	56	62.2	34	37.7	90	36.
2. Chr. endocarditis (benign)	40	85.2	7	14.8	47	18.8
3. Syphilis of aorta	16	36.4	28	63.6	44	17.6
4. Primary myocardial disease (including coronary sclerosis.)	23	76.7	7	23.3	30	12.
5. Chr. lung diseases (rt. heart insufficiency)	20	95.2	1	4.8	21	8.4
6. Bacterial endocarditis	8	88.9	1	11.1	9	3.6
7. Thyroid intoxication	3	50.	3	50.	6	2.4
8. Anemia	1	50.	1	50.	2	0.8
9. Syphilis of heart	...	0.0	1	100.	1	0.4
Total	167	66.8	83	33.2	250	100.0

The lesson of the table for preventive medicine in Baltimore is obvious. I trust it may be sufficiently valid for the Commonwealth of Massachusetts to fulfill the purpose of the founder of these lectures. One third of our myocardial failures are associated with hypertension. As another large group of these patients die of apoplexy, hypertensive cardio-vascular disease assumes first place as a cause of circulatory death. Chronic endocarditis stands next, with syphilis of the aorta just behind, each accounting for about one sixth of the failing hearts. If we could add the deaths due to syphilis of the cerebral arteries, and to ruptured aneurysms, the position of syphilis and of the other infections would be reversed. The clinically primary myocardial insufficiencies, a motley group etiologically, but largely arteriosclerotic, follow with about an eighth of the blame. Emphysema and its congeners are not far behind, then true bacterial endocarditis, then thyroid intoxication, and various minor causes.

CONCLUSIONS.

The following practical conclusions I believe are warranted:

I. Reduction of the mortality from circulatory diseases is attainable.

II. The measures now possible, which will yield definite results, are, in the order of their importance and feasibility:

1. The diminution of syphilitic infection and the early diagnosis and intensive treatment of primary syphilis.

2. The further reduction in preventable infectious diseases, especially diphtheria, scarlet fever and typhoid fever.

3. The education of the public to consider "rheumatism" a serious disease, particularly in childhood, and to seek competent medical advice at once; and the education of the medical profession to treat even the mildest rheumatic fever in bed with large doses of salicylates from the earliest possible moment.

4. The provision of convalescent hospitals for the necessarily protracted after-care of cases of acute inflammatory disease of the heart and of patients recovering from myocardial insufficiency.

5. The development of suitable employments for cardiac patients, and of the social and economic machinery necessary to placing them in such employments.

6. General hygienic measures, including the promotion of temperance.

7. The medical examination of the supposedly healthy at stated intervals.

III. No large reduction of the mortality from circulatory diseases is likely until two groups of problems have been solved:

1. The ultimate causes of hypertension and of chronic nephritis.

2. The infectious agent of rheumatic fever and its portal of entry.

IV. The solution of these problems, if of such a nature as to lead to practical measures, with the elimination of syphilis, would almost abolish disease of the circulatory system, except in the aged.

The need of the moment, therefore, is for more knowledge; not more knowledge of the dangers of circulatory diseases for the public, which means propaganda, but more knowledge of their causes for the physician, which means ceaseless investigation. May I close, in better words than I can master, with Tennyson's lines:

> "Deliver not the tasks of might
> To weakness, neither hide the ray
> From those, not blind, who wait for day,
> Though sitting girt with doubtful light.
>
> . . . . . .
>
> "Not clinging to some ancient saw;
> Not mastered by some modern term;
> Not swift nor slow to change, but firm:
> And in its season bring the law."

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Original Articles.

RESPIRATORY EXCHANGE, WITH A DESCRIPTION OF A RESPIRATION APPARATUS FOR CLINICAL USE.*

By Francis G. Benedict, Boston,
and
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(*Concluded from page* 909)

CRITICAL ANALYSIS OF THE SEVERAL PROCEDURES.

In view of the most searching series of control tests which were made during 1914-15 to discover the reason for the apparently abnormal respiratory quotients found with diabetics, we think it is safe to say that no individual respiration apparatus or, indeed, any type of respiration apparatus has ever been subjected to a more critical or extended study than this. The necessity for demonstrating beyond cavil the accuracy of this apparatus for measuring specifically respiratory quotients makes it important that we should examine critically the individual steps in the process to find if, in spite of the admirable check tests with alcohol and ether, any factors entered into the physiological tests to influence the measurement of either the carbon-dioxide or the oxygen. It should again be stated here that this apparatus must be fitted not only to determine either the carbon dioxide production or the oxygen consumption accurately, but it must determine both factors accurately and thus give an accurate respiratory quotient, or else the service it renders is negligible.

DETERMINATION OF THE CARBON DIOXIDE PRODUCTION.

The determination of the carbon dioxide production by this apparatus is theoretically extremely simple, since it is based solely upon weighing the carbon dioxide absorbing vessels before and after the experimental period. That the entire carbon dioxide production of a man is thus found rests upon two assumptions, first, that the change in weight of the bottles is due solely to the carbon dioxide given off, and second, that the quantities of carbon dioxide remaining in the air inside the chamber are the same at the beginning and end of each period.

In considering the first assumption we should emphasize the fact that the technic must be such as to make sure that the air is dried to the same degree before it enters the carbon dioxide absorbers as it is when it leaves the following Williams bottle or water-absorber. Extensive experience in this laboratory in the past has shown us that errors of this nature are absent with proper attention to the amount of water allowed to accumulate in the Williams bottle.

With regard to the second assumption, namely, the constancy in the amount of carbon dioxide remaining in the air in the chamber, it will be seen that this depends in large measure upon the relationship between the carbon dioxide production and the rate of ventilation of the chamber. With a normal ventilation of 35 to 45 liters per minute and with a regular carbon dioxide production, repeated tests, based upon accurate analyses made in connection with control tests of this apparatus as well as of the bed calorimeter, show that the variations in the residual carbon dioxide in the chamber are practically negligible. If, on the other hand, the subject is more or less restless and there are material differences on the amount of carbon dioxide produced from hour to hour, residual analyses are essential. Complicating the experimental procedure by residual analyses is, however, to be avoided, if possible.

In a large majority of the experiments made in this laboratory, the subject is in complete muscular repose, and comparison experiments should be made only under these conditions. With the subject in complete muscular repose, there is but little variation in the metabolism from hour to hour, and the assumption that the residual carbon dioxide remains constant holds true. Accordingly, if there are changes in the residual carbon dioxide, these are occasioned by such muscular activity as would exclude completely the use of the results for any practical purpose.

It is perhaps especially fortunate, however, that so far as the determination of the respiratory quotient is concerned, material variations in residual carbon dioxide are essentially without effect. If, for example, there is an increment of ½ liter of carbon dioxide inside the chamber during the experimental period, obviously the absorption of carbon dioxide in the soda-lime bottles will be ½ liter too small. On the other hand, the space occupied by the additional carbon dioxide in the chamber would normally have been filled by the admission of ½ liter of oxygen and hence there is a similar deficit of ½ liter in the oxygen measured, the two errors essentially compensating. For all practical purposes, therefore, the measurements of the residual carbon dioxide may be entirely neglected without sacrificing in any way the accuracy in determining the respiratory quotient, even if complete muscular repose is not secured.

DETERMINATION OF THE OXYGEN CONSUMPTION.

While the determination of the carbon dioxide production is very simple in theory and reasonably simple in practice, the determination of the oxygen consumption requires a much more complex procedure. Theoretically nothing is simpler than measuring the oxygen admitted to the chamber as the subject lies breathing normally, but practically the criterion of the amount of oxygen admitted is the position at which the

spirometer bell remains. Thus at the end of an experimental period it is attempted to have the same apparent volume inside the chamber as at the beginning of the period, but even though the apparent volume remains constant, there are several factors which influence this volume. Those of temperature and pressure are obvious, but correction can readily be made for them from the temperature readings and accurate barometer readings provided for. It has already been pointed out in the preceding section that any changes in the carbon dioxide residual in the chamber reduces the oxygen admitted, but it is important again to note that even this substitution of carbon dioxide for oxygen is without material effect upon the respiratory quotient. We have also to consider the possible variations in the measurement of the tension of aqueous vapor inside the chamber.

MEASUREMENT OF THE TENSION OF AQUEOUS VAPOR.

The volume occupied by the water-vapor inside the chamber may fluctuate with the changes in temperature and, indeed, with any changes in the amount of water vaporized from the lungs and skin of the subject, even during repose. These variations are best expressed not in cubic centimeters of water-vapor inside the chamber but, for the purposes of computation, are more advantageously designated as tension of aqueous vapor in millimeters of mercury, the amount of this tension being deducted from the observed readings of the barometer. Instead of determining the tension of aqueous vapor by the gravimetric method, *i.e.* by absorbing water-vapor in U-tubes containing sulphuric acid, we have relied upon an accurate psychrometer comprised of wet and dry bulb thermometers, graduated in 0.1° C. and capable of being read with a lens to 0.01° C. These two thermometers are shown at t_3 and t_4 in the cover of the respiration chamber (Fig. 1, p. 906). By means of the small blower suspended on the inner side of the cover of the chamber, the air is blown with considerable velocity directly over the bulbs of the thermometers. The depression of the temperature of the wet bulb over that of the dry bulb is carefully noted and then, from standard psychrometric tables, the tension of aqueous vapor is computed.

The validity of this method of measuring the water-vapor was tested in several ways, first by introducing a second wet and dry bulb psychrometer in the main ventilating air-current as it left the chamber, and finally by the removal of definite volumes of air and passing them over U-tubes containing sulphuric acid and pumice stone. While it was obviously impossible to secure absolutely the same degree of humidity at each point of measurement, nevertheless when fluctuations occurred, the curves were all parallel and convinced us that the psychrometer as installed gave accurate results. Before each experiment the wet bulb thermometer is removed and thoroughly drenched with distilled water with which its reservoir is filled. Under these conditions most satisfactory results are obtained.

TEMPERATURE MEASUREMENTS.

While theoretically an electrical resistance thermometer with a series of coils distributed in various parts of the cover would be ideal, tests made with a large number of accurate thermometers placed at different points in the chamber showed that average temperature conditions could be obtained by reading the temperatures recorded by the thermometers t_1, t_2, t_4 and t_5. Even with the subject lying inside the chamber, when the small blower on the interior wall is running at a moderate speed, the temperature conditions throughout the chamber, as indicated by the thermometers suspended at different points, seem to be very constant. We have in one case the warm body surface of the subject, which has a temperature of approximately 32° C., and in the other the copper cover, which is in immediate contact with the room air and has a temperature of not far from 20° C. Since in many instances local cooling on the surface of the chamber is necessary, this extraneous temperature may be as low as 18° or even 17° C. In spite of these measurable differences in temperature at different points of the apparatus, all of the thermometer readings show essentially the same curves for temperature fluctuations and we found the average temperature changes in the chamber from the beginning to the end of the period to be accurately given with the four thermometers noted; supplementary thermometers did not in any way alter the values of these differences.

From a rough calculation it can be seen that since the chamber has a capacity of approximately 550 liters, a difference of 1° C. would correspond to an increase in the volume of about 2 liters. As the temperatures are read and the differences computed to 0.01° C., we may therefore assume that the average error for temperature variations is probably not far from ± 20 to 30 c.c. of air. This of course applies directly to the measurement of the oxygen consumed, and is a recognized error in the measurement of this factor. On the other hand, when an experimental period continues for 30 minutes, which is at present the minimum length, this probable error amounts to but 1 c.c. per minute, or about 0.5% of the total oxygen consumption.

MEASUREMENT OF BAROMETRIC PRESSURE.

By the same method of computation as that outlined in the preceding paragraph, it can be seen that each millimeter change in the barometer corresponds to approximately 700 c.c. of air. Accordingly barometric measurements should be made with the very greatest care, preferably to 0.05 millimeter, this corresponding to an error of approximately ± 35 c.c. for each individual period. Obviously the tem-

perature of the barometer with the correction therefor is recorded each time, and from the barometer readings are deducted the value for the tension of aqueous vapor obtained from the readings of the psychrometer as computed from the psychrometric tables.

SUMMARY OF ERRORS IN THE MEASUREMENT OF THE OXYGEN CONSUMPTION.

The measurement of the oxygen consumption is, therefore, subject to the following errors: Errors due to the variations in the residual amounts of carbon dioxide in the chamber at the end of the period; errors due to changes in the temperature and the barometer; and errors in the determination of the tension of aqueous vapor.

As we have already seen, the errors due to variations in the residual amounts of carbon dioxide in the chamber are negligible and play no rôle in the computation of the respiratory quotient, our main objective. The barometer can be read to 0.05 millimeters; taking into account the volume of the chamber (550 liters), this error of reading will correspond to a possible error of ±35 c.c. in the measurement of the oxygen. The thermometers are read and recorded to 0.01° C. as the average of the several thermometers, the possible variation corresponding to a like variation in the oxygen measurement of ± 20 to 30 c.c. Finally the psychrometer is also read to 0.01° C., and with the best psychrometric tables we are assured of an accuracy of but 0.05 millimeters, giving us still another possible error of ± 35 c.c. Should a plus error occur in all three factors, namely, the readings of the barometer, thermometers, and psychrometer, a maximum error of not far from 100 c.c. in the measurement of the oxygen consumption for the period would be conceivable. it is obvious that in actual practice there will be the usual compensation.

It will, therefore, be seen that the longer the experimental period and the greater the amount of oxygen under consideration, the less will be the influence of these errors upon the measurement of the oxygen consumption. And yet, as has been frequently demonstrated in this laboratory, with a subject in complete muscular repose, remarkably regular metabolism measurements, including not only the measurement of the carbon dioxide production and the oxygen consumption, but likewise the determination of the respiratory quotient, may be obtained in one-half hour periods. We may fairly say, therefore, that with a quiet subject results obtained in half-hour periods are perfectly reliable.

CONDITIONS EXISTING INSIDE THE CHAMBER.

As the apparatus is at present designed, the atmospheric conditions with reference to temperature, degree of humidity, air movement, and percentage composition of the air during an experiment are very similar to those obtaining in the chamber of the respiration calorimeter. In other words, the subject is in a relatively confined volume of air which is kept in motion by an electric fan; the temperature is approximately that of room air; the humidity is about 60%, and the pressure obviously that of the barometer. The bed coverings, the exclusion of light save around the head, and the movement of the current of air over the clothed and blanketed body and the uncovered face and hands, appear to have no effect upon the metabolism so far as shown by numerous special tests.

The ideal type of apparatus for studying the respiratory quotient could not be obtained with a respiration chamber for the simple reason that the ideal chamber would be that with a minimum extraneous volume of air. Such ideal conditions would be met, for example, in a diving suit with helmet although, owing to the flexibility of the fabric, the volume of air surrounding the body could never be known. To make a chamber sufficiently small to reduce the extraneous volume of air to a minimum would of necessity require a shape not unlike that of a coffin, and since one of the prime objects in constructing an apparatus for studying the respiratory quotient was to employ it in experiments with diabetics and adapt it for subsequent use in hospital clinics, such an unfavorable shape for the chamber was precluded. The present form, with its volume of 550 liters, is admittedly a compromise.

The most important factor in all respiration experiments is a willing, coöperating subject. With such a subject, the observations are almost invariably successful, but without such a subject, but little of value may be expected. The greatest degree of muscular repose is of prime importance, and it may be considered a general rule that the accuracy of the results is directly proportional to the degree of repose of the subject. Indications of the degree of muscular repose are obtained not from ocular observations, but reliance is placed more particularly upon the records of the pulse-rate and the graphic records of the muscular activity.

SUMMARY OF DESIRABLE FEATURES IN THE CLINICAL RESPIRATION CHAMBER.

The main object in designing this chamber has been to secure an apparatus for determining the *respiratory quotient*, thus necessitating the highest possible degree of accuracy in measuring both the carbon dioxide production and the oxygen consumption. Such a degree of accuracy is, we believe, secured by this chamber, which has the smallest amount of extraneous air volume consistent with the comfort of the subject. Advantageous factors may be considered in two groups: First, as to the subject of the experiment, and second, as to the operator.

ADVANTAGES FOR THE SUBJECT.

The subject lies in a small, well-lighted chamber on a comfortable bed, with no undue restraint of the head, and more particularly of the mouth, lips, and nose by special breathing appliances. The fan inside the chamber keeps the temperature equable and, with the majority of the subjects, produces a distinctly pleasurable environment.

ADVANTAGES IN OPERATING TECHNIC.

Delicate gas analyses are eliminated by this method and the only scientific training needed by the operator is the ability to make simple weighings on a balance and to read the barometer and thermometers accurately. The universal respiration apparatus may be immediately readjusted for observations with either a mouthpiece or nosepieces with a man, or for observations with smaller chambers with infants, dogs, or smaller animals.* The apparatus is capable of perfect temperature control and, with a coöperating subject, periods as short as one-half hour may regularly be obtained.

ROUTINE OF AN EXPERIMENT.

The subject should be lying upon the bed of the respiration chamber not less than 10 minutes, before the cover is put in place. This is especially necessary if the subject has been unusually active or has walked about considerably prior to the experiment. The stethoscope should be attached and records of the pulse-rate begun immediately, as the best index of the quiet condition of the subject is the pulse-rate, which should have practically reached a level before the experiment is begun. The ideal condition for a subject is complete relaxation and muscular repose, with a minimum, regular pulse-rate. The subject may lie either on the back or on the side as, in this type of apparatus, a fixed position of the head is not necessary. Inasmuch as there is always a slight warming of the ventilating blower inside the chamber, due to the passage of the electric current through it, the blower should be started as soon as preparations are begun for the experiment, as the longer the blower runs before the experiment begins, the better will be the results obtained. The subject should be fully dressed, including stockings and shoes; in addition, a pair of woolen socks should be drawn over the shoes, for it has been the experience of most subjects that if any discomfort is felt it is from a sensation of coolness about the feet, ankles, and the lower part of the legs. This is due to the fact that the air inside the chamber is always moderately dry, *i.e.* with a relative humidity of about 60%, and is kept in rapid circulation by the fan blower. The sensitivity of the movable bed may be determined either by pressure with the hand or, more accurately, by dropping a known weight from a certain height upon the bed and noting the amplitude of the marking upon the kymograph record. After the soda-lime bottles and the Williams bottles have been examined to make sure that they have not become exhausted or will not become exhausted before the experiment is over, the cover is lowered with the hand-hole open. It has been found practical to suspend the cover with two cords running through pulleys and to counterpoise it in part to assist in lowering it into place. When the cover is in position, the counterpoise weight is removed so that the cover rests solidly in the water seal on the base of the chamber; the cover of the hand-hole is then put into place.

As it requires some time for the atmospheric conditions inside the chamber to become constant, it has been found advantageous to delay starting the ventilation throughout the air-circuit for approximately 15 minutes. During this time the carbon dioxide accumulates inside the chamber until it amounts to approximately 0.3 or 0.4% of the volume of air. The moisture likewise accumulates to some extent. At the end of 15 minutes the rotary blower is set in motion and the air-current begins to circulate. The several thermometers and the psychrometer are then read. The temperature of the room should be adjusted by opening the doors or windows so as to establish temperature equilibrium as soon as possible, for with a quiet, resting person the difference between the temperature of the room and that in the chamber is approximately 3° C. As soon as temperature equilibrium has been obtained—and this is usually inside of 15 or 20 minutes after the ventilation has been started—temperature readings are taken, a simultaneous reading of the barometer is made, and the position of the spirometer bell is accurately recorded. The valves V_1 and V_2 are then turned.

While the spirometer is so counterpoised as to give 0 in one position, it is not an exact counterpoise, and slight differences occur. It is desirable, therefore, for the operator, while reading the position of the spirometer bell, to hold the counterpoise of the spirometer lightly in the hand in such a manner that the petroleum in the manometer registers exactly 0 at the time that the thermometer is read. If this adjustment is made at the end of each period, the slight differences in the position of the spirometer bell are compensated. From the average temperature readings, the barometer readings, and the record of the position of the spirometer bell, the apparent and real volume of the chamber can be computed, the weight of the subject being taken into consideration, and due allowance made for the displacement of air by the body of the subject.

During the entire time that the motor is run-

* The clinical chamber may also be quickly adapted for studying the respiratory quotient by the Jaquet-Hasselbalch method. In this case the meter required by Jaquet is eliminated and the total carbon dioxide excreted is absorbed by the soda-lime bottles, the air issuing from the chamber being passed through a standard dry gas-meter which by simple computation, permits corrections for the carbon dioxide in the air entering the chamber. This is possible since it is now known that the carbon dioxide of outdoor air, even in relatively congested districts, is constant.

ning, carbon dioxide is being removed from the chamber in both the preliminary period and the main experimental period. This removal of the carbon dioxide causes a diminution in the volume of air in the chamber. In the preliminary period this may be compensated by an increase in the temperature and the spirometer bell will thus remain in essentially the same position as at the beginning or, as is usual, oxygen is introduced to maintain the spirometer bell in a median position. It should be noted, however, that no measurement is necessary of the oxygen introduced during the preliminary period, as only the amount added during the actual experimental period to maintain the spirometer bell at or about its original position is of importance. The amount of air residual in the chamber at the end of each period, as determined by the measurements of the temperature, the barometer, the psychrometer, and the actual height of the spirometer bell, is taken into consideration in the final computations. The manipulation of the universal respiration apparatus has been so frequently and fully discussed that it seems unnecessary to give further details here.

The length of the experimental periods may vary considerably. Theoretically the longer the experimental period is, the more reliable will be the results, for all errors in the readings of the thermometers and the barometer are minimized when extended over a long period. Practically, however, it is very desirable, especially with subjects in the post-absorptive condition, *i.e.*, without food, to shorten the experimental periods as much as possible. Our observers have proved repeatedly that with quiet subjects, especially when they are asleep, determinations with a high degree of accuracy can be made in one-half hour periods. In a large number of cases, after a preliminary period of 35 to 40 minutes, we have been able to secure three successive one-half hour periods with the greatest uniformity in results. If the subjects are restless, and particularly if the restlessness occurs toward the end of the experimental period, the temperature and moisture are materially affected, and the experimental periods must, in consequence, be extended. While it is always a question as to whether it is advisable to attempt any observations with patients as restless and conditions as disturbed as in such cases, yet it has been our almost universal experience that patients, as well as normal individuals, almost always become drowsy during an experiment and are inclined to fall asleep. The air inside the chamber is constantly in motion, the atmosphere is cool and pleasant, there is no odor, and the psychical conditions are, in general, most beneficial and satisfactory. Of practical help in experiments with patients or nervous individuals is a jocular reference to the chamber as a lower berth in a sleeping car; a printed card with absurd "rules" or "regulations" for the occupant may be helpful in dissipating any apprehension during the first hour inside the chamber.

Tables IV, V, and VI show the calculations and summarized results for the period of a typical experiment, including the carbon-dioxide elimination and the oxygen consumption per minute.

CLINICAL USE.

The series of experiments which have been of the most significance in testing this clinical respiration apparatus are those which were carried out in coöperation with Dr. Elliott P. Joslin in the winter of 1914-15 with a number of diabetics. These experiments showed in all cases, save with an extremely irritable boy, who could by no possible means be reconciled to the observations, a most successful use of the apparatus. The subjects were frequently called upon to make dual tests in which the results with the clinical respiration apparatus were compared with those obtained with other forms of respira-

TABLE IV.—CALCULATION OF THE OXYGEN RESIDUAL IN THE CHAMBER AT END OF FIRST PERIOD IN EXPERIMENT WITH MR. C., DEC. 11, 1914.

Psychrometer: Dry bulb (t_1) 20.46° C; wet bulb (t_2) 14.58° C.
Temperature: Head of chamber (t_1) 20.41° C; middle (t_2) 20.44° C; foot (t_3) 19.92° C.
Temperature of apparatus (t_a):

t_1	20.46° C	Avg. (t_a) 20.31° C
t_1	20.41° C	
t_2	20.44° C	
t_3	19.92° C	

Barometer: Reading at end of period (at 19.5° C)	763.75 mm.
Brass scale correction	2.45 mm.
Tension of aqueous vapor in chamber	8.80 mm.
Corrected barometer (p)	752.50 mm.

	Logs.
Residual oxygen:	
Total volume of apparatus .....606[1] liters	= 2.78247
Temperature of apparatus ..... $\frac{1}{1 + 0.00367\ t_a}$	= 9.90877 − 10
Corrected pressure ..... $\frac{p}{760}$	= 9.99569 − 10
Corrected volume $CO_2 + O_2 + N_2$	= 2.74693 = 558.38 liters

[1] The volume of the first chamber used was 606 liters. The volume of 550 liters frequently cited in the text applies to the latest type of chamber.

TABLE V.—CALCULATION OF OXYGEN ADMITTED DURING FIRST PERIOD OF EXPERIMENT WITH MR. C., DEC. 11, 1914.

Spirometer	Start	197.5 mm.
	End	192.0 mm.
	Diff.	5.5 mm.

Correction 5.5 x .023 = + 0.13 liter

Barometer	763.75 mm.
Correction for temperature	2.45 mm.
Correction for aqueous vapor	17.69 mm.
Corrected pressure at meter	743.61 mm.

Meter End	12.76 liters
Meter Start	2.59 "
Diff.	10.17 "
Spirometer correction	+.13 "
Corrected reading	10.30 "
Temperature of meter	20.3° C.

	LOGS.
Meter factor	0.00104
Temperature of meter	9.96879—10
Corrected pressure	9.99053—10
Corrected reading	1.01284
Log. vol. O_2	0.97320
	= 9.40 liter O_2

TABLE VI.—SUMMARY OF MEASUREMENT IN EXPERIMENT WITH MR. C. IN CLINICAL RESPIRATION APPARATUS, DECEMBER 11, 1914.

				Oxygen.						Per Minute.	
Period.	Duration. Mins.	Duration. Secs.	(a) Carbon Dioxide Absorbed. Gms.	(b) Residual at End of Period.[1] Liters.	(c) Change in Oxygen Residual. Liter.	(d) Admitted. Liters.	(e) Consumed (d — c). Liters.	(f) Volume of Carbon Dioxide Produced (a x 0.5091). Liters.	(g) Respiratory Quotient. (f ÷ e).	(h) Carbon Dioxide. C.c.	(i) Oxygen. C.c.
Prelim.	—	—	—	558.61	—	—	—	—	—	—	—
First	50	34	15.43	558.38	—0.23	9.40	9.63	7.86	0.82	155	190
Second	51	13	15.58	558.15	— .23	9.61	9.84	7.93	.81	155	192
Third	50	41	15.32	557.83	— .32	9.61	9.93	7.80	.79	154	196
Average	—	—	—	—	—	—	—	—	0.80[2]	155	193

[1] Residual carbon dioxide + nitrogen + oxygen.
[2] Attention is directed to the extraordinarily high respiratory quotient found with this severe case of diabetes. It is typical of many others following the fasting treatment.

tion apparatus requiring the use of nose- or mouth-breathing appliances. They invariably expressed their preference for the clinical respiration chamber where, as they expressed it, they could lie comfortably and go to sleep. Theoretically, at least, the strain upon the patient during the observation is reduced to a minimum, for he is practically as comfortable as he would be lying upon his bed in the hospital ward, and may take any position desired, the only stipulation made being that he move as little as possible. To assist in the accurate control of the temperature measurements, a signal is given the subject 10 minutes before the end of the experimental period so that he may assume a position which he can comfortably maintain without change for the next 10 minutes. The absence of any abnormality in the conditions is shown by the fact that in a large proportion of cases this signal is not seen by the subject as he is asleep.

In connection with the extensive 1914-15 series of experiments upon the utilization of carbohydrates in diabetes, numerous observations of the respiratory exchange were made with diabetics. The most striking point noted was the frequent appearance of extraordinarily high respiratory quotients,—a phenomenon which at first led us to question seriously the accuracy of the apparatus. In Table VII are given the data for individual periods for a number of subjects prior to January 13, 1915. In Table VIII are given the average values found on nine different days with Mr. Cl. These latter values show that as the fasting continued, the low respiratory quotient, so characteristic of severe cases of diabetes, gradually increased; simultaneously the acidosis disappeared and the total metabolism decreased.

TABLE VII.—METABOLISM OF DIABETIC SUBJECTS IN EXPERIMENTS WITH CLINICAL RESPIRATION APPARATUS.

Name.	Date. 1914.	Carbon Dioxide per Minute. C.c.	Oxygen per Minute. C.c.	Respiratory Quotient.
Miss J.	Oct. 30	124	146	0.85
		131	161	.82
Miss L.	Nov. 4	152	187	.81
		153	186	.83
		154	192	.80
	Nov. 9	146	176	.83
		145	177	.82
		148	187	.79
Mr. C.	Nov. 23	140	172	.82
		142	170	.83
		142	180	.79
	Dec. 2	149	176	.85
		150	176	.86
	Dec. 4	138	170	.81
		137	165	.83
	Dec. 5	135	164	.82
		137	173	.79
	Dec. 9	149	189	.79
		160	200	.80

Name.	Date.	Carbon Dioxide per Minute.	Oxygen per Minute.	Respiratory Quotient.
	Dec. 11	155	190	.82
		155	192	.81
		154	196	.79
	Dec. 17	177	213	.83
		179	220	.81
Mrs. J.....	Dec. 7	156	204	.76
		152	196	.78
Mrs. K.....	Dec. 8	164	222	.74
		143	189	.75
		146	180	.81
Mr. Cd.....	Dec. 16	138	180	.77
		137	183	.75
		140	182	.77
	Dec. 17	121	161	.75
		137	179	.77
Mr. F.....	Dec. 18	199	250	.80
		190	233	.81
		195	250	.78
	1915			
Mr. Fh.....	Jan. 12	213	272	.78
		208	258	.81
		206	254	.81

TABLE VIII.—METABOLISM OF DIABETIC SUBJECT, MR. CL., IN EXPERIMENTS WITH CLINICAL RESPIRATION APPARATUS.

(AVERAGE VALUES PER DAY.)

Date.	Carbon Dioxide per Minute.	Oxygen per Minute.	Respiratory Quotient.
1915.	C.c.	C.c.	
Apr. 15.....	154	215	0.72
16.....	158	217	.73
17.....	152	211	.72
18.....	148	202	.73
19.....	149	195	.76
21.....	140	187	.75
24.....	135	183	.74
27.....	141	189	.75
May 1.....	133	175	.76

The experiments cited in Tables VII and VIII were made at the Nutrition Laboratory, but as a result of the successful outcome of the various tests and the experiments with diabetics in 1914-15, a new clinical respiration apparatus was constructed under the skilful direction of the mechanician of the Nutrition Laboratory, Mr. W. E. Collins which, after long and severe tests, has been installed in the Broadbeck Memorial Cottage of the New England Deaconess Hospital. (See Fig. 3.) The coöperative study on the respiratory exchange of diabetics is now being continued at the Deaconess Hospital by members of the Nutrition Laboratory staff, experiments being made nearly every day.

From the observations in 1914-15 and those now being carried out at the Deaconness Hospital, we are firmly convinced that the clinical respiration apparatus here described makes it possible for hospitals and clinics to secure at a moderate cost a respiration apparatus which will give results with patients that are more consistent and have a higher degree of accuracy than those obtained with any other type of respiration apparatus now used with patholog-

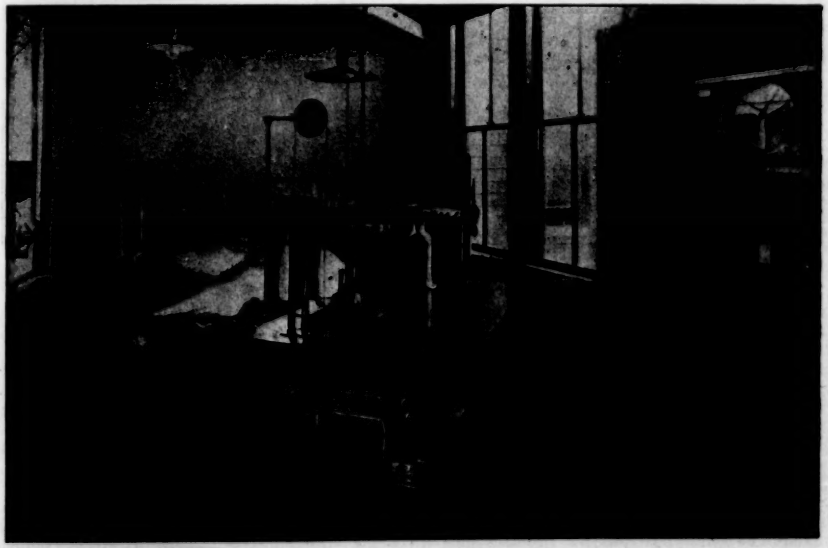

FIG. 3. General view of the complete clinical respiration apparatus installed in the respiration laboratory of the Broadbeck Memorial Cottage, N. E. Deaconness Hospital, Boston, Mass.

Respiration chamber on the left. The thermometers, with reading glass attachments, in the cover; kymograph for registering the muscular activity on the left.

Universal respiration apparatus in the centre. Below are the rotary blower, the motor, and the Williams bottles; above are the spirometer, soda-lime bottles, Williams bottle, and petroleum manometer.

At the extreme right are the barometer, oxygen cylinder, and Bohr meter under water.

ical cases; that it does not involve the time consuming and difficult chemical gas analyses needed for so many other methods; that the technic is readily acquired by young women without special medical, chemical, or physiological training; and that the entire experiment may be made and the results calculated and verified in an unusually short time.

Clinical Department.

DIAGNOSIS OF PERIOSTEAL SARCOMA WITH THE X-RAY.

BY FREDERIC J. COTTON, M.D., F.A.C.S., BOSTON,
Visiting Surgeon, Boston City Hospital.

THE accompanying cut is offered for discussion. For some years, I have noted occasional x-rays of periosteal sarcoma on my own and other services, and in a considerable per cent., have noted the appearance here mentioned. The total series has been too small to be taken very seriously, and I have kept no accurate detail records. For *discussion*, let me put it in this way.

In case of bone thickening, showing in the x-ray plate a picture of slight cortical erosion only, the presence of light stalactite-like threads of bone, perpendicular to the bone surface, strongly suggests sarcoma.

I have not seen this picture in other pathological processes, nor have I seen it noted or commented upon.* If one may conjecture, it seems reasonable that a new-growth, originating on the bone-surface, should, in its rapid growth away from the bone, carry with it certain osteoblasts that produce trabeculae along the *vertical* path of their transit outward. Broadly speaking, periosteal sarcoma (whether it be of round or spindle cell type) does not produce bone to any considerable extent.

The slowly growing granulomata, especially gummata, produce far more bone, but it is laid down (for the most part) in parallel laminations and not in vertical stalactites at all.

The case in point was a boy of 13, who entered with a history of a fall some months previously. For a few weeks he had noticed a lump in the leg; pains were of longer duration. He proved to be a fairly vigorous lad, showing nothing of surgical interest save for the right leg which showed, at the back edge of the tibia, a mass about the length and thickness of the last two phalanges of my little finger; adherent to bone; firm but not hard; non-fluctuant; not more than doubtfully tender; not hot; not pulsating. He had occasional pain in this leg, not very closely localized, and varying from below the knee up to the site of the thickening. That was all there was to go on, save for the x-ray.

I declined to operate, without permission to do a thigh amputation, in order to be ready, in case I got an unfavorable report on the hurry-up frozen section from Dr. Mallory.

The pre-operative diagnosis was periosteal sarcoma. The pathologist said sarcoma (*later* he said lymphoblastoma, but that is only more fashionable—not really different; the popliteal glands were enlarged, but not malignant).

I did a thigh amputation, and so far (too soon to be of any value) all is well.

The question is: did the picture justify the diagnosis, or was there an element of luck?

One more point, and I am done. In this case, *before* I got the specimen, or cut in at all, I had a tourniquet put on near the groin, which remained on till the leg was off. Is there any advantage in this procedure; namely a tourniquet to stop or minimize both blood and lymph flow during manipulation and operation of sarcomata and cancers? If there is, why is it not a routine, as it is not?

Both questions are presented for discussion.

This paper was written in the autumn of 1915. Since then, I have learned that Dr. Walter Dodd of the Massachusetts General Hospital, radiologist, spotted this point in differential diagnosis some years ago, but did not put it on record.

As to the case in hand, the boy died in March of this year of a chest metastasis with repeatedly

* Since writing this, I have seen a more or less similar picture at one or two points in an obvious case of bone syphilis.

recurring massive pleural effusion; so my precautions were too late.

Also his x-rays went astray, and the hospital has not been able to find them. About the time they were lost, I happened to see a specimen in Dr. Mallory's laboratory; a specimen from an amputation by Dr. John T. Bottomley, for a sarcoma of periosteal origin, that developed after a fracture that seems not to have been pathological.

This specimen I had x-rayed, feeling sure that it would show the detail picture above described; and it did. The plate, that appears herewith, is of this specimen and is used by courtesy of Dr. Bottomley.

It represents a later stage than did the lost plate, but it illustrates the point.

I believe this vertical, stalactite formation to be of diagnostic value; I will not call it pathognomonic, but will say this: In any case showing this picture, the burden of proof is shifted; we must show that the lesion is not periosteal sarcoma.

I believe we shall find that it *is sarcoma* in an overwhelming majority of cases in which this curious picture is found.

Therapeutic Medicine.

FURTHER EXPERIENCE IN THE TREATMENT OF INTRACRANIAL HEMORRHAGE IN THE NEWBORN.

BY ROBERT M. GREEN, M.D., BOSTON.

[From the House Clinic of the Boston Lying-in Hospital.]

IN the issue of the JOURNAL for April 30, 1913 (Vol. clxx, page 682), I reported a series of 7 cases of intracranial hemorrhage in the newborn, reviewed the literature on the subject, and discussed the classification of cases, and methods of diagnosis and treatment. Since that time it has been my fortune to observe two further cases of this condition, which seem to illustrate these methods sufficiently well to deserve a special report in detail. I desire herewith to express to the Visiting Physician and the other members of the staff of the Boston Lying-in Hospital my grateful acknowledgment and appreciation of the opportunity to treat and record these cases.

At the time of my previous article I reviewed and summarized the literature on the subject to that date. Since that time the only important reference which I have been able to find in the literature is the work of Moreno[1] on spontaneous and traumatic ruptures of the cranial dura mater in the newborn. He reports a series of forty autopsies on infants dying within a few days of birth in which he found ten cases of rupture of the tentorium and five of the falx cerebri. In all these cases death was due to hemorrhage following the rupture. Moreno's work is well illustrated with a series of seven photographs from the cadaver and is a valuable contribution to the pathology of intracranial hemorrhage of the newborn.

[1] Archives Mensuelles D'Obstétrique et de Gynécologie, April, 1915, p. 145.

The cases which I have to report are as follows:

CASE 1. Baby G. (B. L. H.: 212-57) Female, 8 pounds. Born on March 15, 1915, by multiparous breech extraction, from an S.D.A. position. There was no difficulty with the after-coming head, and the child cried immediately and was of a good color. At 2 p.m. the next day, however, it was found cyanotic and not breathing. There was no evidence of mucus. By hot water immersion and mouth to mouth insufflation it was resuscitated, and appeared as well as ever. During that afternoon it continued to have repeated similar attacks, characterized by apnea, progressive cyanosis, nystagmus, and convulsive twitching of the extremities. I saw the baby that evening with Dr. F. C. Irving. There was no tension of fontanelles or demonstrable alteration of reflexes. The diagnoses considered were cerebral edema, intracranial hemorrhage, and adrenal hemorrhage. There were no localizing signs. Expectant observation was advised.

The baby continued in the same condition throughout March 17. On March 18 it was seen in consultation by Dr. F. B. Talbot, who made a diagnosis of intracranial hemorrhage and advised lumbar puncture, to confirm diagnosis and relieve intracranial tension.

On March 19 the convulsions became more severe and frequent, and lumbar puncture was performed, with removal of 5 c.c. of bloody spinal fluid, following which there was marked remission of the convulsions for 24 hours. When they again became more severe, the puncture was repeated on March 20, and again on the two succeeding days, always with the same result and a marked relief of symptoms. The baby began to take nourishment well (breast milk by medicine dropper) and after March 24 had no further convulsions or respiratory embarrassment. It was discharged well on April 7, nursing at the breast and gaining weight.

On July 16, the patient reported in response to a letter. The baby was well developed, active, gaining weight and appeared in every respect normal.

There seems no reason in this case to doubt the diagnosis, which was confirmed by repeated lumbar puncture. Presumably in this instance the hemorrhage was largely infratentorial, since if there had been any appreciable amount of supratentorial hemorrhage, over the convexity of the cerebral hemispheres, there would hardly have been such notable relief from lumbar puncture alone. It is noteworthy that the symptoms were primarily respiratory,—apnea and cyanosis. At the time of my previous paper I was disposed to feel that clinically cases of intracranial hemorrhage in infants could most conveniently be classified into two groups, according to whether the hemorrhage was chiefly infratentorial or supratentorial. I am convinced, by this and by the following case, that this classification rests on a definite clinical basis of symptomatology and therapeutic indication.

CASE 2. Baby D. (B. L. H.: 215-95.) Male, 9 pounds, 14 ounces. Born on May 7, 1915, by a moderately difficult primiparous forceps extraction from an unrotated O.L.P. position. The baby was resuscitated with difficulty but cried well. There was no important external trauma of the head. Twelve hours after delivery, the baby had a severe clonic convulsion, without associated cyanosis or respiratory disturbance, beginning on the left side and extending less violently to the right. There were two more similar convulsions on May 8. On May 9, the baby refused the breast and began to have more severe convulsions every 15 minutes. At this time the anterior fontanelle, previously soft, became harder and more prominent. There was nystagmus, but no demonstrable alteration of reflexes. The pupils were equally dilated. Lumbar puncture yielded clear cerebrospinal fluid.

In view of this negative puncture, of the primarily left hemiplegic character of the convulsions, and of the absence of respiratory disturbance, there was made a diagnosis of right-sided supratentorial intracranial hemorrhage. After the usual preparation, under light ether anesthesia, an exploratory puncture was done of the right subdural space at the lateral angle of the anterior fontanelle, with the withdrawal of considerable bloody fluid. A 1½ inch incision was then made along the right coronal suture, and the subdural space opened, with demonstration of a diffuse hemorrhage over the convexity of the right cerebral hemisphere. The blood was still fluid or semi-fluid, so that a considerable amount oozed out. A small rubber tissue drain was inserted posteriorly into the subdural space and the skin closed about it with interrupted horse-hair stitches. An exploratory puncture at the left lateral angle of the anterior fontanelle yielded only clear cerebrospinal fluid, so that no further incision was made. A moist boric acid dressing was applied, the head bandaged and the patient put to bed in excellent condition.

Following the operation there was, for a few days, an occasional slight twitching of the left arm, but there were no further convulsions. The baby recovered normally from ether and was fed hourly with breast milk by a medicine dropper. The wick was removed at the end of thirty-six hours, before and for several days after which there was a considerable bloody ooze from the wound. This, however, ceased and the wound healed apparently without infection. There remained a slight bulging at the point of drainage through which cerebral pulsation could be felt. The baby was discharged well and free from any symptoms of cerebral irritation on June 1.

Seen again on October 1, the baby was reported to have continued uninterruptedly well to that date, nursing at the breast and gaining in weight steadily. There remained a slight compressible bulging at the site of operation, but the baby appeared in every other respect entirely normal. There was no alteration of the reflexes and no spastic or flaccid condition of any of the extremities.

This case, in antithesis to the one first reported, presents the typical clinical picture of supratentorial hemorrhage, in which the symptoms were primarily convulsive, rather than respiratory, owing to the irritation of the diffuse hemorrhage over the motor area on the cerebral convexity. The unilateral limitation of the hemorrhage in this instance was quite accurately evidenced by the localizing signs and confirmed by exploratory puncture. It would seem wise that such puncture should always precede operative intervention. The treatment by drainage alone after incision seems, in others' experience and in my own, to afford adequate relief from symptoms due to pressure and to be preferable to an operation involving the removal of bone, which, in these cases, is apparently unnecessary and is very likely to prove fatal from shock.

CONCLUSIONS.

1. Intracranial hemorrhage in the newborn may be most conveniently classified clinically under two groups—infratentorial and supratentorial.

2. In the infratentorial type of hemorrhage the symptoms and signs are primarily respiratory in character and are probably dependent on the pressure of accumulating blood about the respiratory center in the medulla.

3. In the supratentorial type of hemorrhage the symptoms and signs are primarily convulsive and are probably dependent on the irritation of the motor area by accumulating blood over the cerebral convexity.

4. In any case of doubt, diagnosis should be confirmed by exploratory lumbar or cranial puncture, or both.

5. In the infratentorial type of hemorrhage repeated lumbar puncture is probably the best palliative treatment and may prove definitely curative.

6. In the supratentorial type of hemorrhage, the best treatment is incision along the coronal suture at one or both lateral angles of the anterior fontenelle followed by brief drainage with rubber tissue.

7. More extensive procedures than that above outlined are unnecessary and likely to prove fatal.

8. Early diagnosis, and operation within the first two or three days of life, are essential for the best results, since, in such cases, the blood still remains fluid and there is prospect of its complete removal by drainage. The prognosis becomes steadily worse as time progresses, since the clotting of a considerable amount of the blood makes such complete removal impossible, and even if the baby recovers, there is much greater likelihood of subsequent adhesions and irritative Jacksonian phenomena.

LONG ISLAND HOSPITAL TRAINING SCHOOL.—The annual graduation exercises of the Long Island Hospital Training School were held at that institution on June 9. The principal address was delivered by Dr. John H. Cunningham, Jr., of Boston, who presented diplomas to twenty-five pupil candidates, the largest class ever graduated from the School.

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STATE AND FEDERAL NARCOTIC LAWS.

The Harrison anti-narcotic law has now been in operation for more than a year, but will have completed its first annual license period on June 30th. Members of the profession are urged to be prompt in registering and to use the utmost care in complying with all the provisions of the law. This year it will be necessary for an inventory to be made and properly sworn to, which inventory must be sent to the revenue collector, together with the application blank, also properly made out and sworn to.

There is no denying the fact that the provisions of this law entail a considerable amount of work; but we must admit that it was high time that some such law was enacted, and as the rock upon which our profession securely rests its foundation is service to our fellows, we should not only obey the letter of the law, but aid in its enforcement when called upon. It is a matter of regret that Congress did not have the constitutional right to make this a police law, uniform through all the States and administered by federal officers. That was, however, impossible, because unconstitutional; but Congress did have the right to enact a revenue measure and to administer it. The Harrison law purports to be a revenue measure, but a slight study of the situation shows that it is not a revenue-producing act at all, nor was it ever intended to be such.

As a matter of fact, as soon as the Harrison law was in operation, practically all the States hastened to enact laws that should, at least, go as far as the federal provision, and many, like our own State, even went beyond the provisions of the federal law. The point to this is, that the State law is not a revenue measure. It is a police law and is not subject to the same rules in the enforcement of its provisions as is a revenue law. This led to the Supreme Court decision, which was handed down June 5, 1916, and noted in the issue of the Journal for June 15. Mr. Justice Holmes delivered the opinion of the Court, Mr. Justice Hughes and Mr. Justice Pitney dissenting. This is often spoken of as the Chinese possession case.

This was a case where a habitual user had obtained one drachm of morphine, by means of a prescription. The government in this case contended "That Congress gave it (the law) the appearance of a taxing measure, in order to give it a coating of constitutionality, but that it really was a police measure that strained all the powers of the legislature and that Sect. 8 means all it says, taking its words in their plain literal sense." The district judge considered that the act was a revenue act and that the general words "any person" must be confined to the class of persons with whom the act previously had been purporting to deal. To quote further:

"It may be assumed that the statute has a moral end, as well as a revenue, in view—but we are of opinion that the District Court in treating those ends as to be reached only through a revenue measure and within limits of a revenue measure was right."

This decision places the manufacture and sale of drugs in the same position as is occupied by the manufacture and sale of spiritous liquors. The penalty would apply to a person who manufactured and sold without paying his tax, but it would not make a criminal of the customer. The result of this decision will be to confine the

prosecutions to those registered under the Act, and in many cases, at least, that would be where it would naturally fall, for it is quite obvious that the cost and difficulty of an investigation of a considerable portion of our population would be enough in itself to confine it to those who were registered.

At this point, however, comes in our own State legislation which is a police measure and can be administered without difficulty; and the provision of our State law is, that to have narcotics in one's possession without a legal prescription therefor "shall be presumptive evidence that such possession is a violation of this Act." Section 2 provides that a physician shall not give away, prescribe, or deliver any of the prohibited drugs to any person known to such physician to be a habitual user of those drugs, except where the drug is obviously needed for therapeutic purposes.

It will be seen by this that we have an adequate State law, but we are confronted with the menace of a mercenary motive, which is established in Section 1, for there the penalty of violation of this Act is avoided, if the possessor has a legal prescription therefor. To be sure, this is covered in Section 2 by a provision which says that a physician may not prescribe these drugs to a habitual user, except when the drug is obviously needed for therapeutic purposes. Under this law it seems impossible that any man viewing this matter from a strictly business standpoint could, for a moment, undertake to evade the law; for the publicity necessary—in the form of reports—must quickly be in full view of the prosecuting officer; and even if there should be an opening to escape the penalty, through some technicality in a court of law, there is a penalty which is nearly as far-reaching as that provided in the law, and which cannot be escaped by a mere technicality. A man governed solely by a question of policy will not lightly jeopardize his right to practise medicine in the State; and that is precisely what he does, if he fails to fulfil honestly and sincerely both the letter and the spirit of the law. Taking into consideration the prohibition in Section 1 of our State law in regard to possession, and the restrictions in Section 2 imposed upon the prescriber, it would seem as though a great help would be rendered to the prosecuting officer if physicians were required to report to the proper State department all chronic cases requiring narcotics, as well as all cases under treatment for cure of the habit.

The final success of these laws is absolutely beyond question. They have already accomplished a vast amount of good, and their efficiency will increase rapidly during the next few years. They are an epoch-making period in humanitarian legislation, and can only be prevented from a quick realization of their aims by immoral conduct on the part of those legally authorized to prescribe and dispense.

It may be well to call the attention of physicians to the fact that a record-book, distinct and separate from daily cards or case cards, is absolutely necessary in order to comply with the law. A moment's thought will suffice to give the reason. It is a matter of satisfaction to know that in this first year, when rulings and counter-rulings, regulations and counter-regulations have been the order, the medical men, as a rule, have patiently borne the inconveniences imposed upon them by these laws, and in every sense have maintained the ideals of the profession.

THE RÔLE OF THE MEDICAL PROFESSION IN PREPAREDNESS.

THE movement for national military preparedness has in a manner overlooked the important part that the medical profession must necessarily play in any active display of this preparedness. That military medicine is an art quite apart in its organization from civil medicine, and that even a high degree of ability in the practice of medicine does not of itself fit one to practise military medicine, is now well accepted. In countries having compulsory universal military service the medical military arms are provided for on a scale proportionate to the numbers in service. Yet even there in war times it is found that the organized military medical establishments are far from sufficient. Every one of the countries now at war has been glad to avail itself of the help of outside medical units. The training of these units for efficiency for military purposes has not been very difficult, principally because there were in the countries having compulsory service a sufficient number of military medical officers to train the new units into line.

On the other hand, in countries having voluntary military service, on such small scales as here, with the organized medical arms likewise small, it would be a most difficult problem to train civilian medical men for military duty when needed on a large scale and for immediate service. The establishment of the medical reserve corps is a step in the right direction. Its usefulness is somewhat curbed because of the lack of facilities for real training. *The Military Surgeon* of June, 1916, calls attention to the urgent need of organizing the medical profession for this work, either through regular camp work, somewhat after the "Plattsburg Idea," by frequent clinics throughout the country devoted to this subject or by periodic medical society meetings of the same nature. To these suggestions might be subjoined the one of adding to the curricula of the medical schools the subject of military medicine and surgery.

The medical material in this country is numerically very great. The many rural communities have developed physicians who are supremely adapted for emergency medical and surgical work of all kinds such as would be encountered in the field, while the highly specialized medical men of the larger centers would be well adapted for the finer base hospital work. A little extra training and study is all that is necessary to make this material convertible. Besides, even though military medicine is somewhat of a new art, it is an art that must keep abreast with the progress in medicine, and be keen to adopt and adapt it to its particular field. An interest in the military problems of medicine cannot fail to stimulate an interest in the general advances in medicine, which must redound to the efficiency of the profession and to the advancement of the science, as well as to the benefit of the country.

Massachusetts Medical Society.

PROCEEDINGS OF THE SOCIETY.

First Day, June 6, 1916.

Clinics and demonstrations were held during the morning at the principal hospitals in Boston.

The annual meeting of the Supervising Censors was held in the foyer of the Copley-Plaza Hotel, Boston, at 11.30 o'clock. It was attended by fifteen Supervisors. A committee was appointed to arrange for uniform examinations throughout the State, and another committee to consider proposed changes in the rules governing applications for fellowship and the times for holding examinations.

The annual meeting of the Council was held in the same room at noon. (See Proceedings of the Council in the JOURNAL, June 22, 1916.)

Meetings of the Sections were held in the Copley-Plaza Hotel, in the afternoon. The Sections were officered and papers presented according to the following program:

MEETING OF THE SECTION OF MEDICINE.

Foyer, Copley-Plaza, Hotel.
2.30 o'clock.

Officers of the Section of Medicine:

Dr. Henry Jackson, Boston, *Chairman.*
Dr. F. Gorham Brigham, Boston, *Secretary.*
(Program arranged by officers of section.)

Diabetes.

1. Results Obtained in the Treatment of Diabetic Cases, May 1, 1915-May 1, 1916.—Dr. Elliott P. Joslin, Boston.
2. Definition and Determination of Acidosis in Diabetes Mellitus.—Dr. Albert A. Hornor, Boston.
3. Observations on the Blood Sugar in Diabetes Mellitus.—Dr. Orville F. Rogers, Jr., Boston.

Discussion.—Dr. Harry W. Goodall, Boston; Dr. Walter R. Bloor, Boston; Dr. G. Carroll Smith, Boston.

Nephritis.

1. Tests of Renal Function from the Standpoint of the General Practitioner.—Dr. Francis W. Peabody, Boston.
2. Eye Changes in Renal Diseases, Their Diagnostic and Prognostic Value.—Dr. Peter Hunter Thompson, Boston.

Discussion.—Dr. Channing Frothingham, Jr., Boston; Dr. George S. Derby, Boston; Dr. George Van Ness Dearborn, Boston; Dr. H. D. Arnold, Boston.

Attendance, 226.

MEETING OF THE SECTION OF SURGERY.

State Dining-Room, Copley-Plaza Hotel.
2.30 o'clock.

Officers of the Section of Surgery:

Dr. Charles E. Durant, Haverhill, *Chairman.*
Dr. Walter M. Boothby, Boston, *Secretary.*
(Program arranged by officers of section.)

Symposium on Fractures.

1. Fractures of the Base of the Skull.—Dr. Harvey Cushing, Boston.
2. The Importance of Early Reduction of Fractures with Displacement.—Dr. William Darrach, New York City.
3. The Treatment of Hip Fractures.—Dr. Frederic J. Cotton, Boston.
4. Certain Facts Concerning the Operative Treatment of Fractures of the Patella. (With lantern slides.)—Dr. Charles L. Scudder, Boston, and Dr. R. H. Miller, Boston.
5. Some Aspects of the Treatment of Compound Fractures Under Civil and Military Conditions.—Dr. David Cheever, Boston.

Discussion.—Dr. Robert W. Lovett, Boston; Dr. John Bapst Blake, Boston; Dr. John Homans, Boston; Dr. F. B. Lund, Dr. W. J. Mixter, Dr. J. Collins Warren, Boston.

Attendance, 200.

MEETING OF THE SECTION OF TUBERCULOSIS.

Ballroom, Copley-Plaza Hotel.

2.30 o'clock.

Officers of the Section of Tuberculosis:

Dr. Albert C. Getchell, Worcester, *Chairman.*
Dr. John B. Hawes, 2d, Boston, *Secretary.*
(Program arranged by officers of section.)

1. Tuberculosis and Syphilis.—Dr. James A. Lyon, Rutland State Sanatorium.

Discussion.—Dr. W. A. Hinton, Cambridge; Dr. C. Morton Smith, Boston; Dr. R. I. Lee, Cambridge.

2. Tuberculosis Carriers.—Dr. Charles E. Perry, Hampshire County Sanatorium, Haydenville.

Discussion.—Dr. A. K. Stone, Boston; Dr. Adam S. MacKnight, Fall River; Dr. Charles J. Downey, Springfield.

3. What Constitutes Clinical Tuberculosis in Adults. —Dr. George L. Schadt, Springfield.

Discussion.—Dr. Cleaveland Floyd, Boston; Dr. Henry D. Chadwick, Westfield State Sanatorium; Dr. F. P. McCarthy, Dr. Abner Post, Dr. E. O. Otis, Boston.

Attendance, 75.

Officers of the Sections for the ensuing year were elected by the Sections respectively as follows:

Section of Medicine: Chairman, Brace W. Paddock, Pittsfield; Secretary, James H. Means, Boston.

Section of Surgery: Chairman, Frederic J. Cotton, Boston; Secretary, Albert Ehrenfried, Boston.

Section of Tuberculosis: Chairman, Walter G. Phippen, Salem; Secretary, John B. Hawes, 2d, Boston.

The Shattuck Lecture was delivered in the foyer of the Copley-Plaza Hotel, in the evening, by Dr. Theodore C. Janeway, of Baltimore, on "The Etiology of the Diseases of the Circulatory System." Attendance, 457.

Second Day, June 7, 1916.

The Society met at the Copley-Plaza Hotel for the exercises of the one hundred and thirty-fifth anniversary, at 9.30 a.m. The President, Dr. Charles F. Withington, was in the chair, and about 150 Fellows were present during the morning. The minutes were read and accepted. The Secretary announced that during the past year the Society had lost by death 59 Fellows, by resignation 23, by deprivation of the privileges of fellowship 23, making a total loss of 105. The Society had gained 156 Fellows as follows: restoration by the Council, 7, readmitted by the Censors 1, new Fellows 148, a net gain of 51, making the total membership 3633.

The following two proposed amendments to the By-Laws, that had received favorable action at the meetings of the Council, October 6, 1915, and June 6, 1916, and had been published in the call for the meeting, were passed without a dissenting voice:

Section 1 of Chapter II shall be amended by transposing the words "unless otherwise ordered by the Council," in the second and third lines of the said Section, and by omitting all of the text after the word "June" in the fourth line, so that the Section as amended, will read as follows:—

"The annual meetings of the Society, shall be held, unless otherwise ordered by the Council, in Boston, on the second Wednesday in June."

Chapter IV, Section 1.

In the second line after the word, "president" insert the word, "ex-presidents", and in the third line omit the word "and", and after the word "treasurer", insert the words, "and librarian", so that the Section shall read as follows:—

"Section 1. The Council shall consist of councillors chosen by the district societies, and the president, ex-presidents, vice-president, vice-presidents, *ex officiis*, secretary, treasurer and librarian of the general society, and the chairman of each standing committee."

The following proposed amendment to Chapter VII, Section 3, that had received the favorable action of the Council, October 6, 1915, was read by the secretary.

Chapter VII, Section 3, that the word "June" in the last line of the fourth paragraph be changed to "March"; so that the clause shall read: "This dividend shall be apportioned among the district societies according to the number of annual assessments which shall have been paid to the district treasurers previous to March first."

Dr. J. G. Hanson, Treasurer of the Hampshire District Society, asked for the reasons for making the contemplated change in the by-law. These were given by the Treasurer of the Society, Dr. Buckingham, who said that the principal reason was that the methods of collecting assessments were adapted to the requirements of a small society, rather than to one of 3600 Fellows; that it is more business like to have early payment required; that a majority of the disputed bills are those that are paid at the annual meeting; that he did not favor having bills paid at the annual meeting. Dr. Hanson agreed with the Treasurer and thought that some amendment should be incorporated whereby a Fellow could not attend the annual dinner unless his dues were paid in advance of the date of the meeting. Dr. Green, Chairman of the Committee on Membership and Finance, explained that the amendment did not prevent a Fellow from coming to the meeting, paying his dues and receiving his dinner ticket, but it provided that those district treasurers who are active and get in more dues early, receive a larger rebate for their societies. The matter had received considerable attention by his Committee, and it had been introduced by him at the request of his Committee. Two months are a sufficient time, in the opinion of the Commit-

tee, in which to pay dues that are payable in advance, and thus receive the premium of the rebate for so doing.

On being put to a vote, the amendment was passed unanimously.

Dr. J. W. Farlow, Librarian of the Boston Medical Library, reported for a joint committee of the Library and the Massachusetts Medical Society, consisting of H. C. Ernst, J. W. Farlow, E. W. Taylor, G. S. Derby and Channing Frothingham, Jr., to consider the advisability of continuing the index catalogue of the Surgeon-General's Library, at Washington, now that the second series is nearing completion, and offered the following resolution, which was passed unanimously:

Resolved: That the Massachusetts Medical Society accepts the report of the joint committee of the Boston Medical Library and the Massachusetts Medical Society, and strongly recommends that the third series of the index catalogue of the Surgeon-General's Library should be begun and continued to completion.

Dr. Edward Reynolds introduced the subject of the anti-cancer campaign, and explained that it is necessary that the public shall not outstrip the medical profession in its knowledge of or interest in such an important matter. He offered this resolution, which received an affirmative vote.

Resolved: That a committee of five be appointed by the President to consider the duty of the Massachusetts Medical Society towards the education of the members of the profession in this State, in connection with the existing Anti-Cancer Campaign; that they be directed to confer with the American Society for the Control of Cancer through its executive secretary and to report their conclusions at the meeting of the Council in October, 1916.

In accordance with the resolution the President appointed this committee, Edward Reynolds, R. B. Greenough, J. Collins Warren, J. T. Bottomley, E. P. Richardson.

The President made these announcements:

The Surgeon-General of Massachusetts invites the Fellows of the Massachusetts Medical Society to inspect the property of Field Hospital Company No. 1 and Ambulance Company No. 1, at the Commonwealth Armory, head of Pleasant Street, Allston, from 2 p.m. to 5 p.m., Wednesday, June 7.

Dr. Samuel Bates Grubbs, United States Public Health Service, invites the members of the Massachusetts Medical Society to visit the United States Quarantine Station, Gallops Island, Wednesday afternoon, June 7. The quarantine steamer, *Vigilant*, will leave foot of Long Wharf at 3 p.m. and will return at 6 p.m. The methods and equipment for handling plague, cholera, typhus fever and other infectious diseases will be shown, as well as four cases of beri-beri now in the hospital.

Papers were read on the following topics, viewed from the standpoint of public health and preventive medicine:

1. The Relation of the State Department of Health to the Communicable Diseases of Childhood.—Dr. A. J. McLaughlin, Boston.

2. Measles.—Dr. E. H. Place, Boston.

3. Scarlet Fever (Illustrated with lantern slides.)—Dr. F. B. Mallory, Boston.

4. Scarlet Fever.—Dr. C. V. Chapin, Providence, R. I. (Read by title.)

5. Diphtheria.—Dr. J. S. Hitchcock, Northampton.

6. Diphtheria.—Dr. W. H. Park, New York City.

7. Whooping Cough.—Dr. J. L. Morse, Boston.

The Annual Discourse was delivered at twelve o'clock, by Dr. David L. Edsall, of Boston, on "Movements in Medicine." On motion by Dr. F. W. Taylor the thanks of the Society were voted Dr. Edsall for his able and instructive address.

In the afternoon there was a combined meeting of the Sections of Medicine and Surgery at the Peter Bent Brigham Hospital, under the chairmanship of Dr. C. E. Durant, Haverhill, and Dr. Henry Jackson, Boston, and secretaryship of Dr. F. Gorham Brigham, Boston, and Dr. W. M. Boothby, Boston, when the following program was presented.

COMBINED MEETING.

Symposium on Goitre.

1. Medical Treatment of Goitre.—Dr. David L. Edsall, Boston.

2. Surgical Treatment of Goitre.—Dr. C. A. Porter, Boston.

3. Recent Advances in our Knowledge of the Active Constituent in the Thyroid; Its Chemical Nature and Function.—Dr. Edward C. Kendall, Rochester, Minn.

4. Some Recent Experimental Work on the Thyroid Gland.—Dr. Walter B. Cannon, Boston.

5. Metabolism Studies of Thyroid Cases.—Dr. Walter M. Boothby, Boston.

Discussion.—Dr. Frank H. Lahey, Boston; Dr. Malcolm Seymour, Boston; Dr. T. C. Janeway, Baltimore; Dr. M. C. Smith, Lynn.

Attendance, 350.

The Annual Dinner was served in the ballroom of the Copley-Plaza Hotel, in the evening, to 704 Fellows and guests. The President made an address and gracefully introduced the following speakers: Hon. Channing H. Cox, Speaker of the House of Representatives of Massachusetts; Hon. Frederick Lawton, Justice of the Superior Court of the Commonwealth; Rev. George A. Gordon, Pastor of the Old South Church; Professor William T. Sedgwick, of the Massachusetts Institute of Technology; Dr. J. W. Courtney; and the President-elect, Dr. Samuel B. Woodward, of Worcester.

Adjourned.

WALTER L. BURRAGE, *Secretary.*

DEATHS REPORTED FROM JUNE 9, 1915, TO JUNE 7, 1916.

Admitted.	Name.	Place of Death.	Date of Death.	Age.
1893	Bemis, John Merrick	Worcester	Sept. 22, 1915	55
1874	Blood, Robert Allen	Sunapee, N. H.	Feb. 21, 1916	77
1871	Bolles, William Palmer	Santa Barbara, Cal.	Mar. 18, 1916	70
1858	Campbell, Benjamin Franklin	Brookline	Mar. 9, 1916	81
1867	Chase, Horace	Dorchester	Jan. 11, 1916	83
1858	Cheever, David Williams	Boston	Dec. 27, 1915	84
1854	Choate, David	Salem	Apr. 23, 1916	87
1880	Cleaves, James Edwin	Medford	June 20, 1915	61
1890	Darrah, Rufus Elmer	Newport, R. I.	Feb. 8, 1916	54
1872	Dearborn, John George	Charlestown	Jan. 3, 1916	77
1910	Derby, Charles Arthur	New Bedford	Mar. 30, 1914	38
1865	Douglass, John Abbott	Amesbury	Feb. 4, 1916	87
1867	Eveleth, Edward Smith	East Gloucester	Jan. 25, 1916	74
1892	Fahey, James Charles	Northampton	Aug. 24, 1915	48
1873	Fernald, Charles Augustus	Boston	Mar. 15, 1916	68
1892	Follett, Ammi Ward	Somerville	Aug. 15, 1915	57
1868	Foskett, George Mason	Worcester	Feb. 23, 1916	59
1890	Fourtin, Edward Randolph Peaslee	Waltham	Mar 15, 1916	50
1888	Gillard, Arthur Ernest	Lowell	Apr. 15, 1915	50
1893	Goldthwaite, Seth Vale	Allston	Apr. 9, 1916	68
1905	Haché, Henry Clement	Boston	Jan. 7, 1916	47
1914	Hanafin, John Francis	Saranac, N. Y.	May 7, 1916	30
1906	Hancock, Albert William	Salisbury	June 18, 1915	38
1897	Hastings, John Mason	Provincetown	June 10, 1915	45
1880	Hobbs, Ezra Allen	Framingham	Mar. 25, 1916	70
1879	Hooker, Charles Parker	Fortune Rocks, Me.	July 21, 1915	59
1891	Houghton, Silas Arnold	Brookline	Feb. 6, 1916	51
1857	Jeffries, Benjamin Joy	Boston	Nov. 21, 1915	82
1884	Kenney, John Erle	Chelsea	May 3, 1916	54
1879	Kimball, William George	Huntington	Oct. 20, 1915	68
1892	Knowlton, Herbert Eugene	San Diego, Cal.	Oct. 24, 1915	49
1893	Leahy, Thomas Joseph	Cambridge	Dec. 25, 1915	46
1897	Logan, Frank Barker Tays	Gloucester	May 26, 1916	50
1905	Maloney, Thomas Aloysius	New Britain, Conn.	July 13, 1915	40
1889	McCollom, John Hildreth	Boston	June 14, 1915	72
1900	McCormick, Alfred Hugh	Northampton	Oct. 28, 1915	44
1908	Moore, John Francis	Worcester	Apr. 15, 1916	35
1887	Noble, Alfred Ira	Detroit, Mich.	Jan. 20, 1916	58
1891	Noyes, William	Jamaica Plain	Oct. 20, 1915	57
1907	O'Connor, Thomas Hugh	Roxbury	Mar. 30, 1916	59
1862	Pinkham, George Edwin	Lowell	Nov. 15, 1915	75
1873	Redfearn, Joseph	Marlborough	July 1, 1915	66
1888	Richardson, Benjamin Franklin	Lynn	Feb. 15, 1916	52
1912	Roddy, Martin Bernard	Lynn	Aug. 12, 1915	29
1873	Rowe, George Howard Malcolm	Boston	Jan. 30, 1916	72
1871	Russell, Frederick William	Dallas, Tex.	Nov. 20, 1915	71
1883	Stevens, William Stanford	Boston	Apr. 29, 1916	56
1885	Stone, Charles Sinclair	Boston	Apr. 22, 1916	56
1913	Swanson, Axel Fridolf	Easthampton	Mar. 19, 1916	25
1908	Teahan, William John	Holyoke	Jan. 11, 1916	36
1902	Tobey, Edward Nelson	At sea	Aug. 16, 1915	44
1854	Trow, William Marshall	Buckland	Sept. 13, 1913	86
1860	Warren, Orin	West Newbury	Apr. 6, 1916	83
1859	Webster, Joseph Rowe	North Lexington	May 9, 1916	82
1846	Wellington, James Lloyd	Swansea	Feb. 11, 1916	98
1904	Wernick, Benzion G	Roxbury	Feb. 23, 1916	48
1856	White, James Clarke	Boston	Jan. 6, 1916	82
1874	Wight, Daniel Webster	Casco, Me.	Nov. 20, 1915	78
1885	Wolcott, Grace	Heath	Nov. 9, 1915	57

Total, 59.

ADMISSIONS REPORTED FROM JUNE 9, 1915, TO JUNE 7, 1916.

Year of Admission.	Name.	Residence.	Medical College.
1916	Adamian, Hovsep Garo, Lawrence		10
1915	Albee, Kenneth Field, Weston		11
1915	Alcuzar, Isaac, Roxbury		11
1916	Alley, Leon Arthur, Brockton		12
1915	Andrews, Oren, Gardner		7
1916	Armitage, Henry George, Haverhill		12
1916	Bannerman, Walter Bruce, East Bridgewater		9
1915	Barry, Rolla Grant, Worcester		20
1915	Bates, Charles Atwood, Ashburnham		22
1916	Berlin, Maurice George, Dorchester		12
1915	Bone, Herman David, Gardner		22
1915	Brassil, Timothy Francis, Cambridge		12
1915	Brewster, David Truman, Jr., Danvers		15
1915	Brown, Roy Farrington, Fall River		12
1916	Buck, Charles Edward, Boston		12
1915	Bunker, Sidney Moore, Worcester		22
1915	Burt, Clarence Edward, New Bedford		10
1916	Butler, David Mathew, Brockton		12
1916	Butler, Francis Joseph, Worcester		4
1915	Callahan, John Francis, Brockton		12
1916	Carey, Francis Arthur, Taunton		8
1916	Caro, Heiman, Monson		11
1915	Chapin, William Andrew Robertson, Springfield		22
1915	Collins, Frank Laforest, Salem		5
1915	Constans, Frank Elmore, Brockton		21

Year of Admission.	Name.	Residence.	Medical College.
1915	Copeland, Elmer Humphrey,	Northampton	31
1915	Cosby, Edwin Gordon,	South Boston	12
1915	Crandall, Walter Midkiff,	Lawrence	12
1915	Cudworth, Clarence Duane,	Millers Falls	40
1916	Cullen, Charles Andrew,	Hyde Park	12
1915	Curtin, John Francis,	North Abington	32
1916	Cutler, Elliott Carr,	Boston	11
1916	Daniels, Ora George,	Canton	11
1915	Davis, George Rufus,	Marshfield Hills	22
1915	Denny, George Parkman,	Boston	11
1915	Dezell, Frederick Burr,	Lynn	15
1916	Doherty, Francis Joseph,	Brighton	11
1915	Dolan, William Edward,	Worcester	8
1915	Dolan, William Francis,	Somerville	11
1915	Donovan, Sylvester Edward,	New Bedford	29
1916	Doray, Frank Leslie,	Worcester	11
1915	Drury, John Nelson,	Lowell	29
1916	Dutton, Frank Kingsley,	Chicopee Falls	12
1915	Eaton, Harold Burney,	Boston	11
1916	Elkind, Henry Byron,	Worcester	12
1916	Emerson, Paul Waldo,	Boston	11
1915	Farrar, Lonnie Oliver,	Bridgewater	22
1915	Fisher, Gertrude Guild,	Boston	16
1916	Fiske, Eben Winslow,	West Newton	14
1916	Fobes, Howard Edward,	Whitman	12
1915	Forhan, Neil Kittredge,	North Billerica	12
1915	Frasier, Joseph Anthony,	New Bedford	17
1915	Fuller, Solomon Carter,	Westborough	10
1916	Garland, Frederick Eugene,	Gardner	11
1915	Gervais, Harriet Marion,	Boston	12
1915	Gilchrist, John Milton,	Springfield	11
1915	Glidden, Edson William,	Lakeville	32
1916	Golob, Meyer,	Chelsea	24
1916	Golub, Jacob Joshua,	Boston	10
1916	Gould, Carlisle Royal,	Salem	5
1915	Gray, Horace,	Boston	11
1916	Gray, Hugh Barr,	Boston	11
	(Readmitted by Censors.)		
1916	Greaney, William Francis,	Holyoke	4
1916	Green, Harold Russell,	Fitchburg	12
1915	Hall, Charles Francis Adams,	Newburyport	10
1916	Hall, Custis Lee,	Boston	3
1916	Hand, Edward Patrick,	Holyoke	4
1915	Harris, Lorne Wilborne,	Saugus	20
1916	Hassman, David Morris,	Roxbury	12
1915	Hearn, Walter Lawrence,	Lynn	11
1915	Hersam, Norman Paul,	Stoneham	11
1916	Hilliard, William David,	Northampton	20
1916	Hodgkins, Edward Marshall,	Boston	12
1916	Howard, Herbert Handy,	Somerville	11
1916	Hunt, Albert Foster,	Bridgewater	11
1915	Hunt, William Elliot,	Bridgewater	12
1915	Jewett, Everett Porter,	Worcester	12
1915	Johnson, Charles Frederic,	Newburyport	10
1915	Jurist, Charles,	Springfield	29
1915	Kemp, Lysander Schaffer,	Canton	12
1915	Kirkwood, Allan Stewart,	Newton Center	29
1916	Konrad, Frank Charles,	Boston	11
1916	Lane, Elwin Dexter,	Andover	10
1915	La Rivière, Athanase de Charette Evariste,	New Bedford	4
1916	Leavitt, Frank Clyde,	Belmont	19
1916	Leonard, Edward De Witt,	Newton Center	11
1915	Leonard, Zenas Lockwood,	West Stockbridge	29
1915	Levine, Samuel Albert,	Boston	11
1915	Look, Percy Jonathan,	Andover	11
1915	Lowrey, Lawson Gentry,	Danvers	11
1915	Mansfield, Burleigh Burton,	Salem	5
1915	Marsh, Albert,	Reading	15
1916	Martin, David Lorenzo,	Dorchester	10
1916	Marvin, Frank William,	Boston	11
1915	Mathewson, Frank Weeden,	New Bedford	10
1916	Mayers, John Edward,	South Boston	22
1915	Millet, John Alfred Parsons,	Boston	11
1916	McPeake, John Richard,	Mattapan	43
1916	Merrill, Charles Henry,	Lynn	11
1915	Mills, Charles Fisher,	Framingham	11
1915	Miner, Leroy Matthew Simpson,	Newtonville	10
1916	Morriss, William Sarsfield,	Fall River	11
1915	Mott, George Ernest,	Worcester	12
1916	Moulton, Allen Thomas,	Roxbury	32
1915	Nigro, Michele,	Chelsea	11
1916	Orcutt, Wallace Lyman,	West Newbury	2
1916	O'Reilly, Francis Augustine,	Lawrence	12
1916	Papas, Prodromos Nicholas,	Boston	11
1915	Partington, Cyrus Brown,	Jamaica Plain	12
1916	Persky, Myer Arthur,	Pittsfield	12
1916	Pillsbury, Arthur Russell,	Wrentham	22
1916	Powers, William Joseph,	Holyoke	32
1915	Pratt, Emily Adelaide,	Gardner	12
1915	Price, Oscar Jay,	Somerville	12
1916	Prouty, Ira Humphrey,	Marblehead	6
1915	Rapoport, Boris,	Salem	12
1916	Reed, Beatrice Alma,	Taunton	12
1915	Robb, Hunter,	Cambridge	19
1916	Roney, Hugh Beverly,	Pittsfield	21
1915	Schofield, Roger William,	Worcester	11
1916	Schwartz, George Harvey,	Lynn	12
1915	Scott, Norman McLean,	Boston	12
1915	Shapira, Albert Abraham,	Boston	11
1915	Shanks, Charles,	New Bedford	11
1916	Shine, Honoria Kennelly,	Holyoke	33
1916	Silbermann, Maurice,	Revere	35
1915	Skirball, Louis Irving,	Revere	22
1916	Smith, Chiron Waterville,	Marlborough	29
1915	Spinney, Frederic Ira,	Somerville	12
1915	Stansfield, Oliver Holt,	Worcester	10
1916	Steele, George Louis,	West Springfield	22
1915	Stevens, William Russell,	Abington	12
1915	Strong, Seth Lake,	Marshfield Hills	11
1916	Sullivan, Eulick Francis,	Holyoke	22
1915	Sullivan, John Albert,	Pittsfield	15
1915	Talbot, John Edward,	Holliston	11
1916	Tate, Harry John,	Pittsfield	4
1915	Trueman, Nelson Gore,	Danvers	11
1916	Van De Veldé, Honoré,	Boston	38
1915	Walker, Melvin Harvey, Jr.,	Pittsfield	11
1915	Watts, Harry Adelbert,	Malden	21
1915	Wheatley, Frank Edward,	North Abington	11
1915	Wheet, Harry Ray,	Boston	12
1916	Whitcomb, Clarence Adelbert,	Springfield	12
1915	Williams, David Lawrence,	Boston	12
1915	Williams, Frankwood Earl,	Cambridge	13
1916	Williams, Frederick Horace,	Brookline	9
1916	Wright, Willard Lyman,	Springfield	12
1915	Young, Annie Roberts,	Waltham	12

Total, 148+1=149. Restored by Council, 7.

Total gain, 156.

KEY TO MEDICAL COLLEGES.

2 Yale University, Medical Department.
3 George Washington University, Department of Medicine.
4 Georgetown University School of Medicine.
5 Medical School of Maine.
6 Johns Hopkins University, Medical Department.
7 College of Physicians and Surgeons, Baltimore, Md.
8 Baltimore Medical College.
9 University of the South.
10 Boston University School of Medicine.
11 Harvard University Medical School.
12 Tufts College Medical School.
13 University of Michigan, Department of Medicine and Surgery.
14 Dartmouth Medical School.
15 Albany Medical College.
16 Cornell University Medical College.
17 Columbia University College of Physicians and Surgeons.
19 University of Pennsylvania, Department of Medicine.
20 Jefferson Medical College.
21 Hahnemann Medical College and Hospital of Philadelphia.

22 University of Vermont, Medical Department.
24 Medico-Chirurgical College of Philadelphia.
29 University and Bellevue Hospital Medical College.
31 New York Homeopathic Medical College and Flower Hospital.
32 University of Maryland, School of Medicine.
33 Woman's Medical College of Pennsylvania.
35 Medical College of Virginia.
38 University of Louvain, Belgium.
40 Hahnemann Medical College and Hospital of Chicago.
43 College of Physicians and Surgeons, Boston.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY.

Chosen by the Council, June 6, 1916.

Samuel B. Woodward, Worcester, President.
Frederic W. Taylor, Cambridge, Vice-President.
Walter L. Burrage, Boston, Secretary.
Edward M. Buckingham, Boston, Treasurer.
Edwin H. Brigham, Brookline, Librarian.

STANDING COMMITTEES.

For 1916-1917.

Of Arrangements.—E. L. Young, Jr., J. H. Young, J. L. Huntington, R. H. Miller, C. H. Lawrence, Jr., Donald Macomber.

On Publications and Scientific Papers.—G. B. Shattuck, E. W. Taylor, R. B. Osgood, F. T. Lord, R. M. Green.

On Membership and Finance.—C. M. Green, Algernon Coolidge, Jr., Samuel Crowell, F. W. Taylor, Alfred Worcester.

On Ethics and Discipline.—J. A. Gage, J. W. Bartol, Henry Jackson, T. J. Robinson, David Cheever.

On Medical Education and Medical Diplomas.—H. C. Ernst, C. F. Painter, H. W. Newhall, J. F. Burnham, Channing Frothingham, Jr.

On State and National Legislation.—S. B. Woodward, F. G. Wheatley, W. P. Bowers, W. H. Robey, Jr., J. S. Stone.

On Public Health.—M. J. Rosenau, W. I. Clark, Annie L. Hamilton, E. H. Bigelow, R. I. Lee.

PRESIDENTS OF DISTRICT MEDICAL SOCIETIES.

VICE-PRESIDENTS (EX OFFICIIS).

(Arranged according to seniority of fellowship in the Massachusetts Medical Society.)

F. C. Granger, Randolph.....Norfolk South
J. W. Heath, Wakefield.....Middlesex East
W. D. Swan, Cambridge.....Middlesex South
G. O. Ward, Worcester.....Worcester
F. B. Pierce, Haverhill.....Essex North
Emile Poirier, Salem.....Essex South
Paul Thorndike, Boston.....Suffolk
J. V. Meigs, Lowell.....Middlesex North
N. K. Noyes, Duxbury.....Plymouth
G. L. Taylor, Holyoke.....Hampden
A. A. Wheeler, Leominster.....Worcester North
A. C. Lewis, Fall River.....Bristol South
T. F. Greene, Roxbury.....Norfolk
A. K. Boom, Adams.....Berkshire
J. P. Nickerson, West Harwich.....Barnstable
W. H. Allen, Mansfield.....Bristol North
H. N. Howe, Greenfield.....Franklin
C. A. Byrne, Hatfield.....Hampshire

COUNCILORS, 1916-1917.

NOTE.—The initials M.N.C. following the name of a councilor indicate that he is a member of the Nominating Committee. V-P. indicates that a member is a councilor by virtue of his office of president of a district society, and so vice-president of the general society. C. indicates that he is chairman of a standing Committee. E-P. indicates ex-president.

BARNSTABLE,
J. P. Nickerson, V-P., West Harwich.
E. E. Hawes, M.N.C., Hyannis.
C. W. Milliken, Barnstable.

BERKSHIRE,
A. K. Boom, V-P., Adams.
Henry Colt, Pittsfield.
E. A. Kennedy, Great Barrington.
E. W. Markham, Lee.
J. H. Riley, M.N.C., North Adams.

BRISTOL NORTH,
W. H. Allen, V-P., Mansfield.
Sumner Coolidge, Middleborough.
R. D. Dean, Taunton.
F. A. Hubbard, M.N.C., Taunton.
S. D. Presbrey, E-P., Taunton.

BRISTOL SOUTH,
A. C. Lewis, V-P., Fall River.
E. F. Cody, New Bedford.
C. F. Connor, New Bedford.
E. F. Curry, Fall River.
W. A. Dolan, Fall River.
R. W. Jackson, Fall River.
A. H. Mandell, M.N.C., New Bedford.

ESSEX NORTH,
F. B. Pierce, V-P., Haverhill.
G. M. Atwood, Bradford.
R. V. Baketel, Methuen.
I. J. Clarke, Haverhill.
G. E. Kurth, Lawrence.
E. H. Noyes, Newburyport.
J. J. O'Sullivan, Lawrence.
H. P. Robinson, Amesbury.
F. W. Snow, M.N.C., Newburyport.

ESSEX SOUTH,
Emile Poirier, V-P., Salem.
C. H. Bangs, Lynn.
R. E. Bicknell, Swampscott.
N. P. Breed, Lynn.
J. F. Donaldson, Salem.
D. J. Finegan, Gloucester.
H. K. Foster, Peabody.
P. P. Johnson, Beverly.
G. M. Kline, M.N.C., Danvers.
Butler Metzger, Lynn.
P. P. Moore, Gloucester.
W. G. Phippen, Salem.
H. E. Sears, Beverly.

FRANKLIN,
H. N. Howe, V-P., Greenfield.
G. P. Twitchell, M.N.C., Greenfield.
N. P. Wood, Northfield.

HAMPDEN,
G. L. Taylor, V-P., Holyoke.
E. P. Bagg, Jr., Holyoke.
J. M. Birnie, M.N.C., Springfield.
T. S. Bacon, Springfield.
R. S. Benner, Springfield.
E. L. Davis, Springfield.
G. D. Henderson, Holyoke.
M. B. Hodskins, Palmer.
E. A. Knowlton, Holyoke.
A. G. Rice, Springfield.

HAMPSHIRE,
C. A. Byrne, V-P., Hatfield.
O. W. Cobb, M.N.C., Easthampton.
J. S. Hitchcock, Northampton.
M. W. Pearson, Ware.
H. G. Rockwell, Amherst.

MIDDLESEX EAST,
J. W. Heath, V-P., Wakefield.
C. J. Allen, Winchester.
E. C. Fish, M.N.C., Melrose.
W. H. Keleher, Woburn.
G. N. P. Mead, Winchester.

MIDDLESEX NORTH,
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A. E. Bertrand, Lowell.
J. J. Cassidy, Lowell.
J. A. Gage, C., Lowell.
J. H. Lambert, Lowell.
J. J. McCarty, M. N. C., Lowell.
E. J. Welch, Lowell.

MIDDLESEX SOUTH,
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M. H. Bailey, Cambridge.
H. T. Baldwin, Chestnut Hill.
S. O. Baldwin, Framingham.
C. H. Cook, Natick.
H. F. Curtis, Somerville.
E. A. Darling, Cambridge.
D. C. Dow, Cambridge.
A. W. Dudley, Cambridge.
G. W. Gay, E-P., Chestnut Hill.
C. M. Hutchinson, Cambridge.
A. A. Jackson, Everett.
S. F. McKeen, Allston.
G. A. Miles, Somerville.
C. E. Mongan, Somerville.
J. F. O'Brien, Charlestown.
C. E. Prior, Malden.
W. A. Putnam, Cambridge.
Godfrey Ryder, Malden.
Joseph Stanton, Brighton.
E. H. Stevens, M.N.C., Cambridge.
J. O. Tilton, Lexington.
Julia Tolman, Arlington.
G. T. Tuttle, Waverley.
H. P. Walcott, E-P., Cambridge.
C. T. Warner, Marlborough.
G. W. W. Whiting, Somerville.
Alfred Worcester, Waltham.

NORFOLK,
T. F. Greene, V-P., Roxbury.
J. W. Ball, Brookline.
W. W. Barker, Dorchester.
E. H. Brigham, L., Brookline.
A. N. Broughton, Jamaica Plain.
P. W. Carr, Hyde Park.
T. J. Coyne, Roxbury.
A. A. Cushing, Brookline.
W. W. Duckering, Dorchester.
H. C. Ernst, C., Jamaica Plain.
M. H. A. Evans, Jr., Dorchester.
C. B. Faunce, Jamaica Plain.
R. W. Hastings, Brookline.
G. W. Kaan, Brookline.
Bradford Kent, Dorchester.
Joseph Kittredge, Brookline.
W. A. Lane, Milton.
Harry Linenthal, Roxbury.
F. P. McKenna, Jamaica Plain.
T. J. Murphy, M.N.C., Roxbury.
A. P. Perry, Jamaica Plain.
J. W. Pratt, Dedham.
J. A. Reilly, Dorchester.
M. J. Rosenau, C., Brookline.
Victor Safford, Jamaica Plain.
T. M. Shea, Roxbury.
F. W. Sleeper, Dorchester.
R. T. Stearns, Mattapan.
J. L. Sullivan, Roxbury.
A. J. White, Mattapan.

NORFOLK SOUTH,
F. C. Granger, V-P., Randolph.
C. S. Adams, Wollaston.
J. C. Fraser, M.N.C., East Weymouth.
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PLYMOUTH,
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A. A. MacKeen, Whitman.
Gilman Osgood, Rockland.
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F. J. Ripley, Brockton.
F. G. Wheatley, North Abington.

SUFFOLK,
Paul Thorndike, V-P., Boston.
E. S. Boland, South Boston.
H. I. Bowditch, Boston.
G. W. W. Brewster, M.N.C., Boston.
E. M. Buckingham, T., Boston.
W. L. Burrage, S., Boston.
David Cheever, Boston.
H. A. Christian, Boston.
A. L. Chute, Boston.
E. A. Codman, Boston.
J. A. Cogan, Boston.
G. A. Craigin, Boston.
E. G. Cutler, Boston.
R. L. DeNormandie, Boston.
Albert Ehrenfried, Boston.
Channing Frothingham, Jr., Boston.
C. M. Green, C., Boston.
J. B. Hawes, 2d, Boston.
W. C. Howe, Boston.
H. T. Hutchins, Boston.
Henry Jackson, Boston.
R. W. Lovett, Boston.
W. A. Morrison, East Boston.
J. L. Morse, Boston.
Abner Post, Boston.
Anna G. Richardson, Boston.
W. H. Robey, Jr., Boston.
D. D. Scannell, Boston.
G. G. Sears, Boston.
G. B. Shattuck, C., Boston.
G. C. Smith, Boston.
Mary A. Smith, Boston.
Peter M. Smith, Boston.
Richard M. Smith, Boston.
F. B. Talbot, Boston.
H. F. Vickery, Boston.
D. H. Walker, Boston.
John Warren, Boston.
C. F. Withington, E-P., Boston.
E. L. Young, Jr., C., Boston.

WORCESTER,
G. O. Ward, V-P., Worcester.
F. H. Baker, Worcester.
W. P. Bowers, E-P., Clinton.
F. H. Clapp, North Grafton.
W. J. Delahanty, Worcester.
J. T. Duggan, Worcester.
M. F. Fallon, Worcester.
Homer Gage, Worcester.
J. O. Genereux, Webster.
David Harrower, M.N.C., Worcester.
A. G. Hurd, Millbury.
W. L. Johnson, Uxbridge.
F. H. Washburn, Holden.
S. B. Woodward, P., Worcester.
C. D. Wheeler, Worcester.

WORCESTER NORTH,
A. A. Wheeler, V-P., Leominster.
E. L. Fiske, Fitchburg.
A. P. Mason, M.N.C., Fitchburg.
E. A. Sawyer, Gardner.
G. B. Underwood, West Gardner.

CENSORS, 1916–1917.

BARNSTABLE,
E. E. Hawes, Supervisor, Hyannis.
S. H. Sears, Yarmouthport.
C. E. Harris, Hyannis.
S. F. Haskins, Cotuit.
W. D. Kinney, Osterville.

BERKSHIRE,
Henry Colt, Supervisor, Pittsfield.
William Galvin, Blackinton.
H. E. Stockwell, Stockbridge.
G. P. Hunt, Pittsfield.
A. C. England, Pittsfield.

BRISTOL NORTH,
F. A. Hubbard, Supervisor, Taunton.
H. B. Baker, Taunton.
H. G. Ripley, Taunton.
T. F. Clark, Taunton.
T. J. Robinson, Taunton.

BRISTOL SOUTH,
W. A. Dolan, Supervisor, Fall River.
J. G. Hathaway, New Bedford.
C. J. Leary, New Bedford.
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I. N. Tilden, Mattapoisett.

ESSEX NORTH,
J. J. O'Sullivan, Supervisor, Lawrence.
Abbie N. Little, Newburyport.
H. L. Conner, Haverhill.
F. D. McAllister, Methuen.
J. Q. Adams, Amesbury.

ESSEX SOUTH,
N. P. Breed, Supervisor, Lynn.
G. K. Blair, Salem.
O. C. Blair, Lynn.
R. E. Bicknell, Swampscott.
W. V. McDermott, Salem.

FRANKLIN,
G. P. Twitchell, Supervisor, Greenfield.
C. L. Upton, Shelburne Falls.
C. C. Messer, Montague.
E. G. Best, Greenfield.
J. W. Cram, Colrain.

HAMPDEN,
T. S. Bacon, Supervisor, Springfield.
G. L. Woods, Springfield.
W. C. Leary, Springfield.
D. F. Donoghue, Holyoke.
M. W. Harrington, Indian Orchard.

HAMPSHIRE,
M. W. Pearson, Supervisor, Ware.
E. W. Brown, Northampton.
E. D. Williams, Easthampton.
W. J. Collins, Northampton.
J. E. Hayes, Northampton.

MIDDLESEX EAST,
G. N. P. Mead, Supervisor, Winchester.
H. B. Jackson, Melrose.
Ralph Putnam, Winchester.
J. P. Carroll, Woburn.
James Blenkhorn, Stoneham.

MIDDLESEX NORTH,
E. J. Welch, Supervisor, Lowell.
J. P. McAdams, Lowell.
E. J. Clark, Lowell.
G. H. A. Leahy, Lowell.
G. E. Caisse, Lowell.

MIDDLESEX SOUTH,
J. F. O'Brien, Supervisor, Charlestown.
E. S. Abbott, Waverley.
C. E. Hills, South Natick.
F. W. Rice, Brighton.
F. L. Morse, Somerville.

NORFOLK,
R. W. Hastings, Supervisor, Brookline.
W. C. Kite, Milton.
M. J. Cronin, Roxbury.
B. N. Bridgman, Jamaica Plain.
W. J. Walton, Dorchester.

NORFOLK SOUTH,
J. C. Fraser, Supervisor, East Weymouth.
W. A. Drake, North Weymouth.
W. J. McCausland, Quincy.
T. J. Dion, Quincy.
J. A. Peterson, Hingham.

PLYMOUTH,
F. J. Ripley, Supervisor, Brockton.
W. W. Fullerton, Brockton.
J. H. Drohan, Brockton.
Joseph Frame, Rockland.
R. B. Rand, North Abington.

SUFFOLK,
W. H. Robey, Jr., Supervisor, Boston.
C. N. Cutler, Chelsea.
W. C. Howe, Boston.
J. W. Cummin, Boston.
H. W. Goodall, Boston.

WORCESTER,
F. H. Washburn, Supervisor, Holden.
G. E. Emery, Worcester.
W. E. Denning, Worcester.
C. B. Stevens, Worcester.
D. F. O'Connor, Worcester.

WORCESTER NORTH,
A. P. Mason, Supervisor, Fitchburg.
A. P. Lowell, Fitchburg.
W. F. Robie, Baldwinsville.
B. H. Hopkins, Ayer.
H. R. Nye, Leominster.

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1916-1917.

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Berkshire, H. B. Holmes, Adams.
Bristol North, C. S. Holden, Attleborough.
Bristol South, W. E. Synan, Fall River.
Essex North, J. F. Croston, Haverhill.
Essex South, J. E. Simpson, Salem.
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Hampden, F. H. Allen, Holyoke.
Hampshire, J. M. Fay, Northampton.
Middlesex East, E. S. Jack, Melrose.
Middlesex North, F. E. Varney, North Chelmsford.
Middlesex South, L. M. Palmer, Framingham.
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Norfolk South, N. S. Hunting, Quincy.
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Suffolk, F. B. Lund, Boston.
Worcester, R. W. Greene, Worcester.
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1916-1917.

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Hampshire.—C. A. Byrne, Hatfield, President; A. G. Minshall, Northampton, Vice-President; J. D. Collins, Northampton, Secretary; J. G. Hanson, Northampton, Treasurer; F. E. Dow, Northampton, Librarian.

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Middlesex North.—J. V. Meigs, Lowell, President; W. P. Lawler, Lowell, Vice-President; J. A. Mehan, Lowell, Secretary; T. B. Smith, Lowell, Treasurer; P. J. Meehan, Lowell, Librarian.

Middlesex South.—W. D. Swan, Cambridge, President; G. T. Tuttle, Waverley, Vice-President; L. S. Hapgood, Cambridge, Secretary; C. A. Dennett, Arlington, Treasurer.

Norfolk.—T. F. Greene, Roxbury, President; F. W. Sleeper, Dorchester, Vice-President; Bradford Kent, Dorchester, Secretary; G. W. Kaan, Brookline, Treasurer.

Norfolk South.—F. C. Granger, Randolph, President; J. H. Ash, Quincy, Vice-President; F. H. Merriam, South Braintree, Secretary and Treasurer and Librarian.

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Suffolk.—Paul Thorndike, Boston, President; H. F. Vickery, Boston, Vice-President; David Cheever, Boston, Secretary; A. K. Stone, Boston, Treasurer; E. B. Young, Boston, Librarian.

Worcester.—G. O. Ward, Worcester, President; M. F. Fallon, Worcester, Vice-President; E. L. Hunt, Worcester, Secretary; G. O. Ward, Worcester, Treasurer; George Lincoln, Worcester, Librarian.

Worcester North.—A. A. Wheeler, Leominster, President; E. G. Fosgate, Ashburnham, Vice-President; C. H. Jennings, Fitchburg, Secretary; F. H. Thompson, Jr., Fitchburg, Treasurer; L. F. Baker, Fitchburg, Librarian.

MEDICAL NOTES.

PREVENTION OF BLINDNESS IN OHIO.—At the Fourth Annual Convention, National Organization for Public Health Nursing, held at New Orleans May 2, Miss Helena R. Stewart, state supervising nurse under the Department of Public Health Education, State Board of Health, detailed the *modus operandi* of the new law in Ohio. For the period August 20, 1915, to March 31, 1916, 576 cases of inflammation in the eyes of the new-born were reported from 63 out of the 88 counties in that state. Completed records are in file in 351 cases. Physicians reported 196 of these, midwives 101, others 54. It is significant that of the cases reported by physicians, 141, or 72%, used the prophylactic. Out of the 351 cases reported, ten cases of partial blindness were noted.

NEW YORK STATE MEDICAL SOCIETY.—At the recent annual meeting of the New York State Medical Society, Dr. Martin B. Tinker, formerly professor of surgery at the Cornell Medical College, was elected president for the ensuing year.

NEW YORK STATE DEPARTMENT OF HEALTH.—On May 22 Governor Whitman of New York signed an appropriation bill including an item of $65,000 for the purchase of land and the erection of a laboratory building in the city of Albany for the State Department of Health. The site chosen closely adjoins the Albany Hospital and the new Albany Medical School. The laboratory work of the Health Department is at present carried on with great difficulty in an old stable, which has several times been condemned as unsanitary.

RICE MEMORIAL HOSPITAL.—It is announced that Mrs. Isaac L. Rice of New York has purchased from Cornell University a site at Irvington-on-Hudson for the erection of a convalescent hospital as a memorial to her husband, who died in November, 1914.

ENDOWMENT OF CANCER RESEARCH AT COLUMBIA.—The executors of the estate of the late Emil C. Bundy of New York recently paid to Columbia University the sum of $100,000 for the endowment of cancer research.

FIRE IN A CHEMICAL LABORATORY.—Report from Chicago states that on April 21 a small fire with an explosion of gases occurred in one of the buildings of the Abbott Laboratories in that city. This accident was not due to the manufacture of ammunition or explosives, as has been alleged in the public press, whose reports of the extent and character of the accident were much exaggerated. The damage was small and the work of the laboratory was resumed the next morning, as usual.

ENDOWMENT FOR JEFFERSON MEDICAL COLLEGE. A movement has recently been made to secure for the Jefferson Medical College at Philadelphia an additional permanent endowment of $2,000,000. The sum of $100,000 toward this amount has already been subscribed by Mr. David Baugh, founder of the Baugh Institute of Anatomy and Biology, on condition that an equal amount should be raised on or before June 16.

The will of the late Dr. J. William White, trustee of the University of Pennsylvania, and John Rhea Barton emeritus professor of surgery, contained a bequest of $150,000 in trust as a permanent endowment fund, of which the income was to be used for establishing a professorship of surgical research in the medical department of the University, with which the Jefferson Medical School is now affiliated.

WASHINGTON UNIVERSITY MEDICAL SCHOOL.—It is announced that the sum of $1,000,000 has recently become available for the use of the

Washington University Medical School. Of this amount, $350,000 has been given by Edward Mallinckrodt and John T. Milliken of St. Louis, and the remainder by the General Education Board. This sum is to be divided into two funds of $500 each, named respectively after Mr. Mallinckrodt and Mr. Milliken, to be devoted respectively to teaching and research in pediatrics and in medicine, with full-time teachers in this department.

HONORARY DEGREES TO PHYSICIANS.—At the commencement exercises of the various American Universities during the past week it has been noteworthy that relatively few honorary degrees have been conferred on physicians. At Fordham University the honorary degree of Litt.D. was conferred on Dr. James J. Walsh of East Pepperell, Mass. At Washington University, the honorary degree of LL.D. was conferred on Dr. Theobald Smith of the Rockefeller Institute of Medical Research, and at Princeton University the honorary degree of LL.D. was conferred on Dr. Leonard Wood, Major-General of the United States Army. Dr. Theobald Smith also received the honorary degree of Sc.D. from Yale; and Dr. Arthur Dean Bevan, of Chicago, the honorary degree of A.M. At Harvard Commencement President Lowell, in recognition of Dr. Strong's recent services in Serbia, conferred upon him the honorary degree of Sc.D. in the following terms: "Richard Pearson Strong, knight errant of these latter days, armed not like the knights of old, but with the power of science, yet running greater risks than they; destroying dragons invisible to mortal eye, and saving not one or two, but hundreds and thousands by his art."

THE ESTABLISHMENT OF A NATIONAL SCHOOL OF HYGIENE.—Report from Baltimore on June 13 contains the announcement that the Rockefeller Foundation purposes to establish in that city, as an integral part of the Johns Hopkins University, a National School of Hygiene and Public Health.

The immediate need for such an institution, according to the Foundation, has been emphasized by the difficulty cities and towns have had in finding men equipped for promoting public health,—sanitary and preventive measures generally. The possibilities of usefulness of men so equipped, the Foundation pointed out, was demonstrated by the efforts of the American Red Cross in combating typhoid in Serbia and by the International Health Board of the Foundation in its campaign against hookworm disease throughout the world.

It is proposed to erect a building which will cost about $200,000, as one of the Johns Hopkins Hospital group. The running expenses are expected to be about $75,000 annually. The work of organizing the school will be undertaken by Dr. Welch, who is professor of pathology at Johns Hopkins, and Dr. William H. Howell, professor of physiology. It is planned to open the school in October, 1917.

The influence of the school, it is announced, will be extended toward education of the public by exhibits, lectures and other means, with a view to a better appreciation and understanding of the importance of public and personal hygiene and in coöperative efforts for the training of public health nurses.

A NEW VOLUME OF THE OXYRHYNCHUS PAPYRI.—The eleventh volume of the Oxyrhynchus Papyri, now stored at Oxford, England, and recently edited by Drs. B. P. Grenfell and A. S. Hunt, and published by the Egypt Exploration Fund, is reviewed in the issue of the *Lancet* for May 13. The papyrus contains one medical manuscript, a Greek recipe for a cathartic.

"The ingredients do not present any novelty to scholars acquainted with those commonly to be found in the so-called *hiera* (mixtures) of Græco-Roman times. The text runs as follows: 'Ingredients of a purging draught: Cummin 4 drachmæ, fennel 2 dr., parsley 4 dr., costus 4 dr., mastich, 4 dr., coriander 7 dr., 21 laurel berries, nut dr., ham(?) dr., pennyroyal dr., silphium(?), salt, vinegar.' The number of drachmæ for a few of the constituents has become obliterated. The word πέρνα, rendered 'ham,' has probably some other meaning indicating an herb, but a comparison with the lists of ingredients in similar Greek recipes has so far failed to suggest which plant it refers to. The word rendered here silphium is φύλλον, which was a title at this period for silphium from Cyrene. Classic medical authors sometimes used φύλλον to indicate betel-nut. Silphium (asafœtida) is almost certainly the correct interpretation. This papyrus also contains two notes upon the healing art as follows: 'For strangury, to heal the sufferer, take the dry seed of basil-thyme, crumble it with wine of Ascalon, then drink it hot.' The Ascalon wine was celebrated above all the vintages of Syria. The second says: 'For treating wounds, take the fruit of a cypress and boil it and apply.' The remainder of this fragmentary manuscript is occupied with some partly legible portions of what appears to be an apocryphal gospel concerning healing of the sick, not only human sufferers, but angelic ones, the latter being affected with ocular disease."

MANUFACTURE OF RADIUM IN AMERICA.—The United States Bureau of Mines has recently issued a report describing the methods adopted at Denver, Col., in coöperation with the National Radium Institute, for extracting radium and other rare metals from the carnotite ores of Colorado and Utah.

"The plant was first built on an experimental

basis and began production in 1914, but it proved so successful that it was decided to increase its capacity by more than 100%, the construction of the addition being completed in February of last year. The capacity of the plant is 3½ tons of ore a day, and up to last October nearly five grams of radium element had been extracted, and approximately 2½ grams of element delivered in the form of radium bromide, of such a degree of purity as was desired by the hospitals interested in the enterprise. The average cost of one gram of radium element was $37,599 (£7520), though the working costs of the first portion of the plant were much larger than those of the second. In addition, 31,650 pounds of uranium oxide and 11,528 pounds of vanadium oxide were extracted. The method worked out by the Bureau depends on the use of nitric acid, which is recovered as sodium nitrate, and 90% or more of the radium in the ore is extracted. The process has been tried only with American carnotite, and may not be applicable to other radium-bearing ores. The Bureau has been anxious to try pitchblende, but has found it impossible to obtain even a ton for experimental use."

EXAMINATION OF SCHOOL CHILDREN BY PRIVATE PHYSICIANS.—Up to last year, all children attending the public and parochial schools in New York City were physically examined by school medical inspectors of the Health Department's Bureau of Child Hygiene. The large number of children to be examined and the relatively small medical staff available, led the Department to adopt a plan allowing physical examination of school children either by private physicians or by the school medical inspector, at the option of the parent. This plan has now been in operation since last fall, and a brief résumé of the results recorded in the examinations made by private physicians may be of interest.

To date 18,360 cards have been returned, 7,601 in Manhattan, 2,632 in the Bronx, 7,602 in Brooklyn, 373 in Queens and 102 in Richmond. There were 94,426 admissions to schools during the term September, 1915, to February, 1916. (The admission figures for the next term will not be completed until the end of the term.) The cards returned during the first semester up to February 1 totaled 15,606, making a percentage of 16%, which represents the attitude of the parents of the school children toward this procedure, that is, one-sixth of them availed themselves of the opportunity to allow private physicians to do public health work. There was considerable variation in the percentage of cards returned, Brooklyn and the Bronx returning 21% as against 13% from Manhattan and 4% each for the outlying boroughs of Richmond and Queens.

TABLE I.—PRIVATE PHYSICIANS' EXAMINATIONS.

	Number of Admissions.	Examined by Number.	Private Physicians' Percentage.
Manhattan	39,072	5363	13.
Bronx	12,410	2682	21.
Brooklyn	33,963	7186	21.
Queens	6,915	298	4.
Richmond	2,102	87	4.
Total	94,462	15,616	16.

In order to compare the results of the examinations conducted by private physicians with the results obtained by school medical inspectors the following table was prepared, showing the percentage of defects found in 16,203 children examined by private physicians and in over half a million children examined by school medical inspectors.

TABLE II.—PERCENTAGE OF PHYSICAL DEFECTS IN SCHOOL CHILDREN.

(COMPARING RESULTS OF EXAMINATIONS BY DIFFERENT GROUPS OF EXAMINERS.)

	Private Physicians'. 16,203 Examinations.	School Medical Inspection. 1914. 508,595 Examinations.	1915. 278,174 Examinations.
Defective vision.....	3.8	8.9	8.9
" hearing.....	.93	.61	.69
" teeth.....	33.8	50.78	64.67
" nasal breath	11.7	9.35	10.0
Hyp. tonsils.....	20.8	11.72	11.6
Defective nutrition....	10.3	5.24	6.3
Cardiac defects.....	1.9	1.28	1.5
Pulmonary defects....	1.5	.19	.26
Orthopedic defects....	1.5	.60	.72
Nervous defects.....	3.9	.63	.65

Taken as a whole, there is a large degree of correspondence between the two sets of figures. The higher proportion of cardiac, pulmonary, orthopedic and nervous defects found by private physicians is readily understood when one considers the superior surroundings and greater time at the disposal of private physicians making these examinations in their offices.

If the 15,616 examinations had been conducted by the Department of Health they would have required the services of school medical inspectors and nurses as follows:

School medical inspectors	at $1200 per yr.,	4	=$4800
Nurses.....	at 900 per yr.,	1½	= 1350
			$6150

As it was, they cost the Department approximately $700 for circulars of information, examination cards, postage, etc., this constituting a saving to the Department of $5,450.

POLIOMYELITIS IN NEW YORK CITY.—Within the past month an unusual number of cases of poliomyelitis have been reported from Brooklyn. Altogether, some two dozen cases have been reported since June first, all but one in the practice of private physicians.

According to the Health Department investigation, the cases are mild in character and have

so far occurred principally in infants and very young children. All but one of the cases are in private families and in no instances have two cases occurred in one family.

The Department requires a minimum period of quarantine of six weeks and also insists on the exclusion from school of other children in the same family. The disease is probably spread through infected nasal discharge and the Department, therefore, insists on precautions similar to those in diphtheria.

The Department of Health calls attention to the fact that diagnosis of suspected cases is aided greatly by an examination of the cerebrospinal fluid. Such examinations are made free of charge by the Health Department's laboratory. Physicians trained in the diagnosis of the disease will consult with attending physicians on request.

An extensive outbreak of infantile paralysis embracing over 2,500 cases occurred in New York in 1907 and was carefully studied by a special committee appointed by the New York Academy of Medicine.

In order to make certain that all cases of infantile paralysis are being reported, a house-to-house canvass is to be made of each block in which there is known to be a case of poliomyelitis. Every family in such a block will be visited by a nurse attached to the Bureau of Preventable Diseases, who will endeavor to find any additional suspicious cases of the disease. If she finds that such a case is under the care of a private physician, she will at once report that fact to headquarters, so that the Department of Health may take up with the physician the proper isolation, etc., to be pursued. If no physician is in attendance, a competent diagnostician will at once be detailed by the Department of Health to visit the patient and take whatever steps are necessary to prevent the spread of the disease to others.

Acting Commissioner Billings has addressed a letter to every Brooklyn physician, calling attention to the existence of poliomyelitis in Brooklyn and asking for their coöperation in controlling the disease.

Seven additional cases of poliomyelitis were reported from Brooklyn on June 20, two of them in one family. At the Health Department, the increased number reported was thought to be largely due to the newspaper publicity of the past few days. Further increase in reported cases is expected within the next few days as the result of the 2500 circular letters sent out on June 19 by Acting Commissioner Billings, to all the physicians in Brooklyn. Most of the cases thus far reported have occurred among Italians. The youngest patient in the group reported June 20 is nine months old. Three of the patients are two years old, one is three years, one four years, and another five years old. The house-to-house canvass now being conducted has not yet been finished. As far as can be determined, the disease is practically localized in the old section of Brooklyn.

Inasmuch as only ten new cases suspected to be infantile paralysis were reported to the Department of Health in response to the personal letter sent to each Brooklyn physician, the health officials in charge of the investigation expressed the view that the outbreak was not spreading and that conditions would probably soon return to normal. Investigation of some of the suspected cases yesterday led to the inclusion of four additional true cases, making the total number in Brooklyn in the present outbreak 47, of which three have ended fatally. The Health Department expects to complete its investigation of all the suspected cases by this evening and will at once prepare a list of true cases under observation.

PREVALENCE OF MEASLES IN WASHINGTON.—The weekly report of the United States Public Health Service for June 16, states that during the week ended June 3, 1916, 409 cases of measles with one death were reported in Seattle, Wash. The epidemic of measles began in this city on February 15, 1916, since which time there have been in Seattle a total of 3921 cases of the disease, with eight deaths.

EUROPEAN WAR NOTES.

MEDICAL ASPECTS OF THE DUBLIN REVOLT.—In the issue of the JOURNAL for June 8, we commented on certain medical aspects of the Dublin revolt of April 24. The issue of the *Lancet* for May 13 contains a further statement of the casualties and of the hospital and ambulance arrangements. These details are of particular interest to those to whom Dublin is familiar as a medical center.

The official figures announce the casualties as 124 killed and 388 wounded. Irish daily papers of the date of May 6 contain the names of 160 civilians killed, probably a total of 200 is a conservative estimate. Somewhat less than this number of soldiers were killed.

"Of wounds, Jervis Street Hospital, which is in the center of Dublin, close to the General Post Office, treated some 400 cases. Mercer's Hospital, which is close to the Royal College of Surgeons, treated 130 cases of shot wounds. The City of Dublin Hospital and Sir Patrick Dun's Hospital, which are near the Northumberland Road and Mount Street Bridge area, also treated large numbers, and all the city hospitals, as well as the Dublin Castle Red Cross Hospital and the King George V. Military Hospital, received their share. The total may have been anything between 1,000 and 2,000. The great majority were, of course, civilians, including a large number of women and children. This is, perhaps, inevitable in street fighting, especially when the locality is strange to both troops and officers. The presence of a few rebels, or even of one sniper in a particular house or court, endangered the lives of several score innocent per-

sons, and in many cases those innocent suffered for the guilty. Civilians were also shot by the rebels, chiefly in the first hours of the outbreak.

"As the arms varied greatly, so also did the character of the wounds. The wounds produced by military rifles were such as the war has accustomed surgeons to, but the number of head wounds was notably large. Most of these were received by civilians who incautiously looked through their windows while the military were being fired at by rebel snipers in their neighborhood. The firearms possessed by the rebels seem to have varied within the widest limits. Many of them apparently had serviceable rifles, such as Mausers and Winchesters, but all sorts of arms were used. Many round bullets have been picked up, and it is probable that they were fired from shotguns, the bullets being substituted for shot in the cartridges. It is said that the rebels were possessed of a few machine-guns.

"It is too soon to judge of the effects of treatment, but one may hope that the results will be better than have been generally seen in military operations in France. In the first place, treatment was given speedily, and in the second the clothes of the wounded were fairly clean, and were not, as in France, sodden with infective mud.

"Some of the hospitals which gave most attention to the wounded have already been mentioned, but as fighting was taking place simultaneously in several quarters of Dublin, all the city hospitals were kept busy. In some instances the wounded could not, on account of danger in the street, gain access to the general hospitals, and the special hospitals gave accommodation willingly. In particular, the National Maternity Hospital in Holles Street received a great many civilian patients. The Rotunda Hospital, which was for some time occupied by the troops, also treated casualties and received dead bodies, while the Royal Victoria Eye and Ear Hospital took overflow patients from several institutions.

"Two urgency hospitals were opened by the local branches of the British Red Cross Society and the St. John Ambulance Association, one in Merrion Square with 20 beds, and one in Lower FitzWilliam Street with 25. They served as overflow hospitals for Sir Patrick Dun's and Mercer's Hospitals respectively. The staffs of the several hospitals worked under great difficulties. In some cases surgeons were unable to reach their own hospitals at all, and in most cases those who were able to get to their hospitals had perforce to stay there for several days. The members of the staffs of the Adelaide, Jervis Street, the Mater Misericordiæ, and the Richmond Hospitals, for instance, were virtual prisoners in these institutions from Easter Monday or Tuesday to Saturday or Sunday. Many of the hospitals were repeatedly struck by bullets, but I have not heard of any casualty in a city hospital. For a night and a day Jervis Street Hospital was in imminent danger of fire, as the great Sackville Street fire steadily approached it by the two lanes of Henry Street and Abbey Street. The same hospital was threatened by the fire of a big gun, the officer in charge of which was unaware that the building was a hospital. The Adelaide Hospital, close to Messrs. Jacobs's factory, a rebel stronghold, had to stand some rounds from a machine-gun intended for Jacobs's. As the city gas-supply was cut off early in the week operative work in the hospitals was rendered very difficult.

"The work of transporting the wounded to hospital was performed with much courage. Royal Army Medical Corps ambulances plied to the Castle and the King George V. Hospitals.

"Considerable damage was done by fire to public buildings, especially the Post Office and the Imperial Hotel. The building of the Royal College of Surgeons, which was one of the first to be occupied by the insurgents, fortunately received but slight damage. The windows overlooking St. Stephen's Green were riddled by rifle and machine gun fire, which also did some damage to the walls and furniture. Some of the Library books had been employed to form barricades."

War Relief Funds.—On June 24 the totals of the principal New England relief funds for the European War reached the following amounts:

Belgian Fund	$128,449.26
Allied Fund	122,053.82
Serbian Fund	100,495.72
French Wounded Fund	89,998.38
Army Huts Fund	59,480.60
French Orphanage Fund	56,315.23
Armenian Fund	50,648.28
Surgical Dressings Fund	36,512.67
Facial Hospital Fund	22,357.55
Italian Fund	20,093.56
Cardinal Mercier Fund	6,892.50
Artists' Fund	2,532.62

BOSTON AND NEW ENGLAND.

The Week's Death Rate in Boston.—During the week ending June 24, 1916, there were 233 deaths reported, with a rate of 15.98 per 1,000 population, as compared with 192 and a rate of 13.38 for the corresponding week of last year. There were 34 deaths under 1 year, as compared with 30 last year, and 81 deaths over 60 years of age, against 53 last year.

During the week the number of cases of principal reportable diseases were: diphtheria, 54; scarlet fever, 20; typhoid fever, 1; measles, 320; whooping cough, 48; tuberculosis, 46.

Included in the above were the following cases of non-residents: diphtheria, 11; scarlet fever, 5; measles, 2; whooping cough, 1.

Total deaths from these diseases were: diph-

theria, 3; measles, 6; tuberculosis, 20; whooping cough, 1.

Included in the above were the following deaths of non-residents: diphtheria, 1; tuberculosis, 2.

BOSTON CITY HOSPITAL ALUMNI FIELD DAY.—The annual reunion and field day of the Boston City Hospital Alumni and house officers was held on the new South Department grounds, West Roxbury, on Tuesday, June 27. The athletic program, in charge of Dr. Paul Withington and Dr. John Woodside, began at three o'clock by a baseball game between the alumni and the house officers, followed by races, swimming events and other games. Supper was served at six o'clock.

DEBARMENT OF MILK FROM BOSTON MARKET.—Report from Rutland, Vt., on June 16 states that as the result of a recent inspection of local farms by inspectors of the Boston Board of Health, nearly forty dairies in Rutland, Bennington and Windham Counties have been forbidden to ship milk, butter or cheese to the Boston market until they have complied with various requirements of the inspectors for making changes in their barns and dairy accessories.

DISCONTINUANCE OF CAMBRIDGE MILK STATIONS.—It is announced that during the coming summer the system of milk stations which has been maintained for several years in Cambridge will be discontinued. In its stead two graduate nurses trained in infant welfare work will visit the homes of babies throughout the city. It is expected that this method will reach a larger number of patients and will help families to help themselves in the care of their children better than the system of milk stations.

MAINE HOMEOPATHIC MEDICAL SOCIETY.—The fiftieth annual convention of the Maine Homeopathic Medical Society was held at Augusta, Me., on June 13. The speakers included Dr. John A. Hayward of Portland on "History of the Maine Homeopathic Society," and Dr. John P. Sutherland, dean of the Boston University School of Medicine, who gave an address on "Hahnemann's Homeopathy."

The following officers were elected: President, Dr. L. A. Brown of Portland; vice-presidents, Dr. F. A. Ferguson of Portland and Dr. W. H. Walters of Fairfield; treasurer, Dr. W. S. Thompson of Augusta; corresponding secretary, Dr. Carrie E. Newton of Brewer; chairman of the board of censors, Dr. H. F. Marin of Bath.

OPENING OF THE BURRAGE HOSPITAL.—The Burrage Hospital on Bumpkin Island, in Boston Harbor, was reopened for its fourteenth season on Thursday, June 8.

"The association which maintains this charity is incorporated under the laws of Massachusetts, with Mr. Burrage as president of the corporation; Mrs. Alice H. Burrage, treasurer, and Albert C. Burrage, Jr., clerk. Dr. Philip A. Shinn, a graduate of Tufts Medical School of last summer, who has since been on the staff of the Robert B. Brigham Hospital, has been engaged as resident physician. The surgeons are Dr. W. F. Wesselhoeft, Dr. T. E. Chandler and Dr. C. T. Howard. Dr. O. W. Chadwell is in charge of the medical department; Dr. George H. Earl is in charge of the orthopedics, and Dr. Carlisle Reed is the examining physician.

"The consulting surgeons are as follows: Dr. Charles F. Painter, dean of Tufts Medical School and chief of staff of Robert Brigham Hospital; Dr. Lloyd T. Brown of the Massachusetts General Hospital and the Robert Brigham Hospital; Dr. Mark Rogers, Dr. Robert B. Osgood and Dr. Harry C. Low, all of the Massachusetts General Hospital; Dr. Charles H. Lawrence of the Robert Brigham Hospital, Dr. E. P. Richardson, surgeon at Robert Brigham Hospital and his assistant surgeon, Dr. Richard H. Miller, who is connected also with the Massachusetts General; and Dr. Edward N. Libby of the City Hospital."

Bumpkin Island was originally granted by royal charter of King James of England to Samuel Ward, who on his death bequeathed it to Harvard College, from whom it is now leased by Mr. Albert C. Burrage.

"The hospital is in the form of the letter H, 175 feet long by 153 wide, and sets just back of the crown of the island, which contains fifty acres. It is two stories high at the front and three at the back, of light buff brick with staircases of iron to make it absolutely fireproof. In the central section, extending from the rear, is an inclined runway which, by long slopes, leads from floor to floor for the convenience of wheel chairs and the ambulance. On the first floor are the administrative offices and reception rooms, the dining rooms, kitchen, serving rooms and scullery, all large enough to provide for the utmost capacity of the building. There are four large wards on each floor, and each contains fourteen beds and smaller adjoining wards. There are a number of bathrooms. The operating room is equipped with all appliances known to modern surgery.

"On the second floor are rooms where special patients or critical cases may be placed for more careful observation. There are convalescent rooms and a library, as well. On the upper floor of the central wing are nurses' and servants' quarters with laundry, drying rooms and ample storage place in the large airy basement. All the walls of the hospital are of faced brick, enameled in white. The building is supplied with water piped from Hingham. There are gas and steam plants on the island."

The institution is essentially a convalescent hospital at which the preference is given to orthopedic cases. Only patients between the ages of two and fifteen and with non-contagious diseases are admitted.

INCIDENCE OF COMMUNICABLE DISEASE IN CHELSEA, MASS.—The annual report of the Board of Health of Chelsea, Mass., for the year 1915, states that a total of 988 cases of reportable diseases were recorded. This increase of 202 from last report is accounted for by the large number of cases of measles occurring in November and December. Diphtheria showed a decrease from 107 cases in 1914 to 70 in 1915; scarlet fever a decrease from 222 to 78; typhoid fever a decrease from 37 to 35; measles showed a great increase—from 194 to 462; 293 of these cases occurring in November and December. Ophthalmia neonatorum showed an increase from 28 to 36. Many of the cases were very mild inflammations, and some were apparently no more than an irritation following the use of the silver nitrate solution. One case of pellagra was reported from the United States Marine Hospital. There were 127 cases of all forms of tuberculosis reported. The deaths numbered 29, the least number since 1900.

HARVARD DENTAL ALUMNI ASSOCIATION.—The forty-fifth annual meeting and dinner of the Harvard Dental Alumni Association were held in Boston on June 21. The principal addresses were made by Dr. Eugene H. Smith, dean of the Harvard Dental School, and by Dr. Charles A. Brackett. The following officers were elected for the ensuing year: president, Dr. Ernest A. Chute; secretary, Dr. Charles T. Warner; treasurer, Dr. James J. O'Brien.

MASSACHUSETTS STATE NURSES ASSOCIATION.—The thirteenth annual meeting of the Massachusetts State Nurses Association was held in Boston on Tuesday of last week, June 13. The address of welcome was given by the president, Miss Sara E. Parsons.

"At 11 in the morning the League of Nursing Education met at 636 Boylston street, and had as speakers Miss Helen Wood, R.N., Miss Ellen T. Emerson and Miss M. E. P. Davis, R.N. A meeting for the organization of the section for private duty nurses was held at 585 Boylston street, at 1 in the afternoon. The councillors met at the same place at 2.30, after which followed the business meeting. Speakers included Representative John J. Kearney of the public health committee of the Massachusetts State Legisature, on 'Value of State Control of Training Schools for Nurses,' and David C. Gibson, R.N., on 'The Place of the Graduate Male Nurse in the Profession.' Discussions followed talks by Miss Ellen T. Emerson of Concord, Miss Emma M. Nichols, R.N., Miss Carrie M. Hall, R.N., and Miss Esther Dart, R.N. The committee on arrangements included Miss M. E. P. Davis, R.N., chairman, Miss Sara E. Parsons, R.N., and Miss Charlotte W. Dana, R.N."

The following officers were elected for the ensuing year: President, Miss Sara E. Parsons; vice-president, Miss Lucia L. Jaquith; second vice-president, Miss Mary A. Meyers; recording secretary, Miss Julia A. Smith; corresponding secretary, Miss M. E. P. Davis; treasurer, Miss Esther Dart; historian, Miss Mary M. Riddle.

INVESTIGATION OF NON-PULMONARY TUBERCULOSIS.—Among the resolves (Chapt. 62) of the current session of the Massachusetts General Court may be noted the following, with reference to the investigation by the State Department of Health of non-pulmonary tuberculosis.

"*Resolved*, That the state department of health is hereby authorized and directed to investigate the matter of non-pulmonary tuberculosis with special reference to children and adolescents throughout the commonwealth. The department shall determine so far as possible:—

"First, the present number of cases of non-pulmonary tuberculosis in the commonwealth, and their situation.

"Second, the number of hospital beds now available for the care of such cases.

"Third, the number of additional hospital beds needed for the proper care and treatment of such cases, and the proper situation of such beds.

"Fourth, whether such additional beds, if needed, should be supplied by additions to, or enlargements of, existing general or other hospitals, or by providing new institutions designed for the purpose of treating non-pulmonary tuberculous cases exclusively.

"Fifth, how such additions to or enlargements of general or other hospitals, or such new institutions, if they are needed, should be financed and administered.

"The said department may hold such public or private hearings as it may deem proper for the purpose aforesaid, and shall report to the next general court, on or before the second Wednesday in January, with its conclusions and such recommendations and drafts of proposed legislation as it may deem expedient. To carry out the purposes of this resolve, the department may expend a sum not exceeding five hundred dollars."

This resolve was officially approved on April 27, 1915.

MASSACHUSETTS COMMISSION FOR THE BLIND.—The recently published annual report of the Massachusetts Commission for the Blind states that of 40,259 school children examined last year in the cities of Cambridge, Lynn and New Bedford, 1,892 or 4.7 per cent. have only one-half normal vision or less. Of these children, 3.1 per cent. have never previously had their eyes examined or been provided with glasses. The important problem arises as to how many of these children should be relieved entirely of regular school work on account of their defective vision.

"The facts brought out in this year's study more than confirm the estimate, presented in our last report, that at least .1 per cent. of school children require special education in defective eyesight, or conservation of eyesight, classes. In the three cities studied this year at least 68, or .17 per cent. of the 40,259 public school children should be transferred to such special classes. It is, therefore, probably safe to estimate that fully .17 per cent. of the 382,548 school children in cities, or at least 650, require such classes. As for the 193,962 children in towns, .17 per cent., or at least 330, may be expected to need special training.

"The commission reported that during the twelve months ending Nov. 30, 1915, only one new case of total blindness from ophthalmia neonatorum was discovered in this State. The commission said that there might be other cases not unknown, but that it was safe to say that the menace from this cause of blindness has been reduced to less than 1 per cent. as a factor in bringing about loss of sight."

VENERABLE LIVING HARVARD PHYSICIANS.—The recently published annual necrology of Harvard University shows that an exceptionally large proportion of the oldest living graduates are physicians. The senior alumnus of the college is Dr. Nicholas Emery Soule of Exeter, N. H., who was born in 1824, and received the degree of A.B. in 1845. The only survivor of the class of 1846 is also a physician, Dr. Abner Little Merrill of Boston. The senior alumnus of the Lawrence Scientific School is Dr. William Lewis Jones of Atlanta, Ga., who received the degree of S.B. in 1851. The class of 1843 of the Harvard Medical School has two survivors, Dr. Kimball Hill and Dr. Edward Philippe LeProhon.

Miscellany.

CHANGES IN COST OF DRUGS.

DURING the past quarter there have been various modifications in the cost of standard drugs as affected by war conditions, sometimes resulting in an increase, and sometimes in a decrease in price. Report from New York on April 13, describes in part as follows, the condition of the drug market at that date.

"Fluctuations still are the rule in the market for drugs and chemicals. Supplies in the majority of cases are at a lower point than ever, despite all attempts at domestic production. Quicksilver and all mercury products, carbolic acid, hydrogen peroxide, quinine, chloride of lime and such alkali products as caustic soda and soda ash are the only ones which have shown tendency to decline.

"Corrosive sublimate, powdered and granular, has been reduced 50 cents, to $2.23 and $2.28 per pound; calomel 45 cents, to $2.53 per pound; mercury bisulphate 55 cents to $2.19 per pound; red precipitate, powdered and granular, 45 cents to $2.83 and $2.93 per pound; white precipitate 50 cents to $2.93 and $2.98 for the powdered and granular; blue pills, mass and powdered, 20 cents to $1.35 and $1.37 per pound; mercury with chalk, 20 cents to $1.37 and $1.39 per pound; mercurial ointment "½" 35 cents to $1.43 and $1.44 per pound; mercury "1-3" $1.38 and $1.39 per pound; and citrine ointment 20 cents to 80 and 81 cents per pound.

"Sensational advances have been the rule in citric as well as in tartaric acid, tartar emetic and cream of tartar. For citric acid $1.00 to $1.10 per pound is asked against a maker's contract figure of 64 and 64½ cents per pound. Tartaric acid is now quoted at approximately 85 and 88 cents for the crystals and 82 cents for the powdered.

"Agar-agar or Japanese isinglass, also used as a gelatine, has advanced to 40 and 70 cents per pound, while antimony oxide is nominal at 50 cents and antimony powdered needle to 45 and 47½ cents per pound. Russian cantharides have been held up by the closing of the port of Archangel and prices have advanced to $8.50 and $9.00 per pound. But oil of coriander, another product of Russian origin, which has been held at $50.00 per pound, has dropped to $30.00 per pound from $35.00 per pound. So far as Japanese menthol is concerned, demand has been comparatively light, and local sellers have continued to quote the comparatively low figure of $3.15 per pound in cases and $3.20 per pound in broken lots. The high cost of tea siftings as well as the heavy demand which has brought stocks of caffeine alkaloid in this country practically to the vanishing point has resulted in an advance to $20.00 per pound for the alkaloid and $9.75 per pound for the citrated. Oil of anise also had advanced to $1.10 and $1.15 per pound, following a rise in "star" anise seed to 25½ and 26½ cents per pound.

"All offerings of ichthyol have been withdrawn by the controlling factors, owing to their inability to procure further supplies from Mexico. Substitutes are being offered at $6.50 against a price of $4.50 for the original product. It is understood that Tecolutla, the only shipping port now remaining open in Mexico, has been practically closed to merchandise shipments.

"The market for all coal tar products continues to pursue an extremely anomalous course. Carbolic acid has undergone several successive declines to a basis of $1.08 and $1.10 per pound in drums and $1.19 to $1.22 per pound in pound bottles while forward contracts for deliveries late this year as well as during 1917 have been booked as low as 80 and 85 cents per pound The domestic output has been greatly increased of late and is now estimated at from

800 to 1000 tons per month. The only sympathetic decline which is noted with carbolic acid is in aniline oil, contract prices of which have been reduced to 68 and 72 cents per pound on deliveries commencing April 1, and running for the balance of this year and the first half of 1917. The spot market shows little relaxation, however, due to the tremendous demand for this material in the dye field. A pronounced upward movement has taken place in practically all other coal tar medicinal drugs and among the new levels attained are acetanilid $2.75 per pound; antipyrene $55.00 and $60.00; coumarin $9.00 and $10.00 per pound; phenolphthalein $22 per pound; saccharine $13.00 and $13.50 per pound; benzoate of soda $6.00 and $6.50 per pound; benzoic acid from toluol $7.00 per pound; and vanillin 57 and 60 cents per ounce. The salicylates have been somewhat easier, mainly owing to a falling off in demand in England where production is on the increase. The spot quotations for salicylic acid have declined to $3.95 and $4.00 per pound against a previous quotation of $4.25 and $4.50 per pound, while salicylate of soda has declined to $3.90 and $4.25 per pound.

"Norwegian cod liver oil has soared to new heights notwithstanding considerable improvement in the yield. As there was no carry-over from the 1915 crop and as the demand is in excess of the output, prices have advanced to $140 and $175 per barrel. It is understood that the great bulk of the Newfoundland output is being disposed of to England and that prices for this oil have advanced to $120 and $125 per barrel. A noteworthy advance also has taken place in Harlem oil to $2.75 per gross bottles, following cablegrams advising that Dutch producers have raised their prices. For castor oil there are still no offers of any importance in the local market below 28 and 30 cents per pound, but forward offers of the number 3 oil have been noted from England at 20 cents per pound.

"The market for narcotic drugs has ruled comparatively steady under the moderate general demand from the trade and a fair export demand for morphine and codine from Europe and Latin America.

"American manufacturers of quinine continue to hold their output strongly on the basis of 75 cents an ounce. There is no weakness in this drug despite the fact that fairly large blocks have been consigned here from Holland and England."

On April 20 there was a further reduction in the cost of mercurials, and other chemicals fluctuated in different directions.

"Sugar of milk was advanced by one leading maker to 20 cents for bulk in boxes. Quinine was firm, a fair export demand having been noted. Aspirin was quoted at 88 to 90 cents an ounce in second hands. Resorcin was shaded to $19 and $29 per pound. An out-of-town house advanced santonine crystals to $37, and powdered to $38. Castor oil declined to 21 and 22 cents per pound for 'A.A.' Offers of benzoate of soda were made somewhat more freely at $5.50 and $5.60 per pound. Weak holdings of chlorate of potash were eliminated, and the market was firmer at 72 and 75 cents. Benzoate of soda was obtainable at concessions, $5.50 and $5.60 per pound being freely quoted."

On April 22 there were further changes in market prices which were reported from New York in part as follows on that date:

"Notwithstanding lack of activity there were a number of important price alterations in the drug and chemical market on Friday, the most conspicuous feature of the market being a lowering of salicylic acid to $3.75 and $4.00 per pound, following freer offers. A further easing off in prices for a great many of the leading staples was noted. Citric acid was steadier with offers as low as 85 cents in some quarters. Outside lots of tartaric acid become available at 80 cents per pound. Japanese camphor also reacted somewhat following larger arrivals and slabs were obtainable at 51 1-2 and 52 cents and tablets at 53 and 53 1-2 cents. Chloroform eased off to the extent that offers appeared at 65 cents per pound. The market for antipyrene was reported lower, though some sellers continued to ask $55 against $45 quoted in other quarters of the trade. Other products in which the tendency was downward were domestic aniline oil and salts, carbolic acid, alum, potash, lump and powdered, epsom salts and methyl salicylate, which is obtainable on nearby contracts at $2.25 per pound.

"Among the products in which the undertone is considered stronger are dynamite and refined glycerine, benzoic, boracic, pyrogallic and oxalic acids, amyl acetate, caffeine alkaloid, formaldehyde, Norwegian cod liver oil, bay rum, which is held at a minimum of $1.70 per pound in barrels; arsenic, gum assafœtida, orange and bergamot oils, quicksilver, saccharine, benzoate of soda, sugar of milk, Venice turpentine and Japanese vegetable wax.

"Some makers were delivering limited supplies of salicylic acid on contract at $2.25 per pound, and the spot market was easier at $3.75 and $4.00. The scarcity of benzoic and boracic acid was more pronounced and the small lots obtainable were held at higher premiums.

"The bulk of this year's output of Norwegian cod liver oil has been sold to Germany and the small lots remaining were held at $200 per barrel in the primary market. The essential oils were featured by a much stronger tone in bergamot and orange, but lemon was steady, despite the reports that prices are being forced higher in Messsina.

"There was considerably more buying, particularly in a small lot way, of quicksilver at the decline to $125 per flask, and it was believed that the bottom had been touched for the time

being. Mercurials were unchanged. The market for opium was lifeless with the gum maintained on the basis of $11.50 per pound in cases and the powdered and granular grades at $113 per pound. Codeine and morphine met with a moderate demand at makers' quotations.

"Acetanilid was offered at $2.30 and $2.35; acetphenetidin at $25 and $26; antipyrene was in better supply and nominally lower; chloral hydrate was also reported somewhat easier. Creosote beechwood was quoted down to $8.50 per pound, a decline of 50 cents. The scarcity of saccharine and sugar of milk was pronounced and prices were strong. Bromine was offered as low as $3.00 for technical. The market for benzoate of soda was extremely strong and $5.50 was inside."

Report from New York on April 26 noted few changes from conditions of the previous fortnight.

"The general situation surrounding all coal tar medicinal products shows no noteworthy relaxation for the reason that makers are still unable to obtain any quantity of the necessary intermediaries. Among the features of the market yesterday was a sharp advance in Japanese oil or camphor to 20 cents per pound following the advance in the gum. Ceylon citronella oil was advanced by some sellers to 53 1-2 cents per pound. Botanical drugs were featured by an advance in Russian powdered cantharides to $8.50 and $9.00 per pound, an advance in angostura bark to 40 cents and an advance in echinacea root to 20 and 21 cents following large sales at those prices.

"Cablegrams received yesterday from Yokohama stated that quotations for menthol for prompt shipment from Japan had advanced to 11s. 6d. per pound, equivalent to $3.35 per pound spot New York. Menthol was still obtainable at $3.15 and $3.20 per pound in cases in the local market, but higher prices were expected in view of the higher freights on the Pacific. Salicylic acid was weaker at $3.75 and $3.90 per pound."

On May 2 it was reported from London that the British government has purchased the entire output of Norwegian cod liver oil for the coming season.

Further report from New York on May 4 noted a sudden recent decline in certain drug prices:

"Mercurial manufacturers announce a sharp decline of 20 to 40 cents per pound in all hard and soft preparations in consequence of the easier position of quicksilver, which is being offered at $115 per flask of 75 pounds. Trading in the general list of drugs and chemicals was reported quieter, as far as domestic demand was concerned. Export demand, however, is reviving, and one of the bright spots yesterday was the much firmer position of chemically pure glycerine following sales to Italy of 200 cases or 6000 pounds. Formaldehyde also is in wide export demand, an order from England for one carload per month (May—December, inclusive) involving more than $26,000, having been closed. One of the outstanding features of the situation yesterday was the weakness in salicylate of soda, spot offerings of 2000 pounds being reported from one holder at $3.45 per pound, a concession of 45 cents over previous quotations. Holders of bichromate of potash are reported to be realizing and 64 cents was quoted in one quarter. Other sellers maintained prices for bichromate of potash at 69 and 70 cents and bichromate of soda at 50 and 53 cents.

"Silver nitrate touched the highest point on the current movement yesterday when prices were advanced 2 3-8 cents an ounce to a minimum of 47 3-4 cents. Offers of bromide of soda appeared at $3.40 per pound, a concession of 10 cents, but second hands were asking higher premiums for bromide of potash and strontium. Salol was reported easier at $9.25 per pound for May delivery. Boric acid was firm with offerings of spot goods from 14 1-2 to 17 cents per pound."

Further report from New York, on May 23, notes other fluctuations in the drug market as follows:

"There were several other weak spots in the market, offers of salicylates being reported as low as $2.75 and $3.00 per pound, which is a concession of 50 to 75 cents per pound. A sharp reduction was named in nitrate of silver, prices being 1⅝ cents an ounce lower, at 45¼ and 47¼ cents an ounce. An easing off in prices throughout the list of potash preparations carried bicarbonate of potash to $1.40 per pound, against $1.65; permanganate to $1.65 and $1.75, yellow prussiate to $1.55 and $1.60 and red prussiate to $4.75. Sugar of lead and sugar of milk still are extremely scarce under heavy demand. Saccharine also is in good demand and there are orders in the market for large quantities at $14.00 and $14.50 per pound."

Further report from New York, on June 1, states that on that date there was a sudden rise in the cost of Norwegian cod liver oil from $150 to $175 a barrel, and on Newfoundland cod liver oil from $135 to $140.

"This advance follows cablegrams from Norway stating that the fishing season has just terminated and that of the total output of 47,600 barrels of oil this season, Germany had taken 42,000 barrels. The advance in the Newfoundland oil was based on the belief that England was about either to commandeer all stocks or place an effectual embargo on all future shipments, to guarantee a supply for home consumption. It is now an open question as to how long further supplies of the Newfoundland oil will be forthcoming, in view of the fact that England is today in dire need of this oil. Manufacturers of emulsions and other medicines who have always

depended upon Norwegian cod liver oils as their base products have had to resort to Newfoundland cod liver oil, which has consequently advanced, as above stated."

Further report from New York, on June 6, states that there has been a further reduction in the cost of glycerin to 55 cents a pound.

"One of the significant features of the market is that many of the declines which have taken place have been on comparatively small sized offers. Thus, while it is possible to buy genuine aspirin in small lots at 70 cents, a round lot of 1000 ounces could hardly be picked up at less than 75 cents, and the same situation is true in caffeine, salol, quinine, benzoate of soda, coumarin and tartaric and citric acids. Offers of hydroquinone are now quoted at $6.00, a concession of 50 cents, and benzoate of soda is also obtainable at a 50 cents concession at $5.50. Menthol also has declined 10 cents, and is now offered at $2.90. Salvarsan continues scarce, and is offered only in small lots at $10."

The scarcity of West Indian sandalwood oil has increased the price from $3.50 to $3.75 per pound.

TUBERCULOSIS IN BOSTON.

The Boston Health Department has recently published a survey of conditions in this city with regard to the incidence, prevalence and mortality of tuberculosis. The following table presents a summary of the incidence and mortality of the disease during the past fifteen years:

Year.	Population.	Cases Reported.	Deaths.	Deaths per 10,000.	Cases Unreported until Deaths.
1900...	561,892	*815	1,407	25.06	...
1901...	568,763	1,149	1,348	23.67	...
1902...	576,049	944	1,247	21.65	...
1903...	583,335	1,355	1,227	21.08	...
1904...	590,621	2,138	1,280	21.67	695
1905...	597,908	2,168	1,224	20.47	605
1906...	613,075	2,131	1,185	19.33	406
1907...	628,242	2,623	1,138	18.11	481
1908...	643,409	2,646	1,094	17.00	400
1909...	658,576	2,905	1,072	16.28	320
1910...	673,045	3,479	1,163	17.28	328
1911...	684,811	3,137	1,067	15.58	62
1912...	712,534	3,332	1,093	15.34	40
1913...	724,493	3,195	1,064	14.69	46
1914...	736,442	2,645	1,045	14.19	40
1915...	748,431	2,775	1,034	13.82	27

* Seven and one-half months.

The above figures show a continuous decline in the incidence of tuberculosis since the Boston Health Department began its work of conflict against the infection in 1900.

"In 1900, with a population of 561,892, there were 1407 deaths from pulmonary and laryngeal tuberculosis. In 1915, with 748,341 inhabitants, the deaths were 1072. This is a decline from 25.00 per 10,000 in 1900 to 13.82 in 1915.

"In 1915 there were 81 non-residents who died of tuberculosis in Boston, while 181 residents of Boston died in other places in Massachusetts, chiefly in State institutions. More residents of Boston go to hospitals in other places in the State for treatment for tuberculosis than come in from outside.

"In May, 1900, the Boston Board of Health first began to require that all cases of pulmonary and laryngeal tuberculosis should be reported. The idea was new and progress was slow. The medical profession did not heed the request readily. Hesitancy in making a diagnosis or regard for the patients' feelings caused physicians to delay notification of the disease, or to neglect to report entirely. For the first four years the reported cases were less than the number of deaths. Efforts were made to correct this imperfect registration. Physicians were requested to give their reasons for failure to report. On Aug. 1, 1907, the State Board of Health declared tuberculosis to be a disease dangerous to the public health, thus making it a criminal offence for a physician or householder to fail to report. More rigorous methods were then taken. Every death from tuberculosis that was not found in the registry of reported cases was investigated, and warning was given that delinquents would be prosecuted. Flagrant offenders were brought into court, and a number fined $50 each for failure to comply with the law. A great improvement resulted. The unreported deaths fell from 481 in 1907 to 62 in 1911, and in 1915 these had been reduced to 27. Apparently 97% of all the cases are reported. Evidently the fear of punishment accomplished what gentler measures could not do. The few cases still unreported come mostly from hospitals, where the disease is discovered only at time of death or from autopsy findings.

"Of the deaths that occur annually in Boston from tuberculosis, 67% have not been reported for more than one year; 17% for not more than two years; 11% for not more than three years. The Department, for this reason, has made a general survey of all the cases in its registry up to Jan. 1, 1916, and there were 12,200 names on file. Only 5,100 were definitely located.

"The number of pulmonary and laryngeal tuberculosis cases in this city is probably not more than 7,000, and certainly not less than 6,000. The number of cases is at least six times the number of the annual deaths from the disease. Owing to the difficulty in locating the patients, no more accurate estimate can be made at present.

"The conditions of tuberculosis in Boston show much improvement. It is a great satisfaction to state that not a single neglected, bedridden case was found in the city. Most of the patients who would have come under this class had gone voluntarily to hospitals. Others had been removed to hospitals by order of the health department because their presence at home was a danger. At the present time practically all the

advanced cases that cannot be cared for at home can find suitable hospital accommodations. If the whole campaign against tuberculosis had accomplished no more than this, it would have well repaid the efforts expended.

"As to hospitals, the Boston Consumptives' Hospital has 410 beds, sixty of them for children; the trustees of the hospital are authorized also to hire 100 beds in private hospitals. At Long Island there are seventy-five. This gives 585 beds under city control. In addition, there are 131 in private tuberculosis hospitals. In the State hospitals at Rutland, Westfield, Lakeville, North Reading, there are 1000 beds, and in the State institution at Tewksbury, 425, giving in all, accommodations for 1425 patients. To these hospitals, Boston sends its share of patients.

"It has been estimated that as many available beds as there are deaths annually from this disease should be the minimum standard for real progress in tuberculosis work. This figure has almost been reached when the available beds in the city and State art considered."

From these data the Department derives the following conclusions, which are commended to the attention of physicians throughout the Commonwealth:

"1. That the deaths from pulmonary and laryngeal tuberculosis have been slowly but steadily decreasing in this city.

"2. That the cases are well reported.

"3. That hospital accommodations are satisfactory.

"4. That there are no neglected cases.

"5. That the general knowledge of tuberculosis has much improved, and cases treated at home are cared for in an intelligent manner."

NOTICES.

United States Navy Medical Corps.

The next examination for appointment in the Medical Corps of the Navy will be held on or about August 7, 1916, at Washington, D. C.; Boston, Mass.; New York, N. Y.; Philadelphia, Pa.; Norfolk, Va.; Charleston, S. C.; Great Lakes (Chicago), Ill.; Mare Island, Cal.; and Puget Sound, Wash. Applicants must be citizens of the United States and must submit satisfactory evidence of preliminary education and medical education. The first stage of the examination is for appointment as assistant surgeon in the Medical Reserve Corps, and embraces the following subjects: (a) anatomy, (b) physiology, (c) materia medica and therapeutics, (d) general medicine, (e) general surgery, (f) obstetrics. The successful candidate then attends the course of instruction at the Naval Medical School, which will begin on or about October 1, 1916. During this course he receives a salary of $2000 per annum, with allowances for quarters, heat, and light, and at the end of the course, if he successfully passes an examination in the subjects taught in the school, he is commissioned an assistant surgeon in the Navy to fill a vacancy. Full information with regard to the physical and professional examinations, with instructions how to submit formal application, may be obtained by addressing the Surgeon General of the Navy, Navy Department, Washington, D. C.

W. C. Braisted,
Surgeon General, U. S. Navy.

Changes in the Medical Corps, U. S. Navy, for the Week Ending June 17, 1916.

June 12, Assistant Surgeon M. R. C., H. A. Royster, commissioned from May 15, 1916.

June 13, Surgeon F. L. Benton, detached Naval Station, Key West, Fla., to duty with expeditionary forces, Santo Domingo.

P. A. Surgeon W. J. Zalesky, to Naval Recruiting Station, Brooklyn, N. Y.

Assistant Surgeon V. H. Carson, detached Navy Recruiting Station, New York, N. Y., to duty with expeditionary forces, Santo Domingo.

June 17, Assistant Surgeon John Harper, detached Navy Yard, New York, N. Y., to *Minnesota*.

Assistant Surgeon Arthur Freeman, to Navy Yard, New York, N. Y.

Worcester Patriotic Parade.—The physicians of Worcester and vicinity are invited to march in a body in the patriotic parade to be held in that city on July 4, 1916. It is hoped that each hospital will be represented by a float or by marchers or by both, thus making a medical division. Every physician taking part in this parade is to wear a straw hat, a white band with a green cross on the sleeve and carry a small flag. All who are willing to participate in this demonstration should immediately notify the secretary of the undersigned committee. Sleeve bands and flags will be ordered in accordance with these notifications. Announcement of further details will be published in the daily papers.

Committee, Sam'l B. Woodward, Homer Gage, John T. Duggan, M. F. Fallon, J. K. Warren, Fred'k H. Baker, Ernest L. Hunt, *Secretary*

Wanted: Medical Men for the Service in London. There is need for 40 men to serve in the London war hospitals. They are needed for six months' service. Transportation and board and lodging will be provided by the British government. Recent graduates in medicine can be used, as well as men with longer experience, as general practitioners, internists, or surgeons. For further information apply to Dr. R. C. Cabot, 1 Marlborough Street, Boston, Mass., or Dr. H. A. Christian, Peter Bent Brigham Hospital, Boston, Mass.

APPOINTMENTS.

Harvard Medical School.—The following physicians have been appointed associates for the ensuing academic year:

Farrar Cobb, A.M., M.D. (Surgery).
Frederic Jay Cotton, A.M., M.D. (Surgery).
William Edward Faulkner, A.B., M.D. (Surgery).
Joshua Clapp Hubbard, A.B., M.D. (Surgery).
Daniel Fiske Jones, A.B., M.D. (Surgery).
Fred Bates Lund, A.M., M.D. (Surgery).
Abraham Myerson, M.D. (Neuropathology).

RECENT DEATHS.

Dr. Ellis V. Fanning, who died at Nantucket, Mass., on June 16, was a graduate of the Harvard Dental School in 1901 and had practised his profession at Brockton, Mass., since that time. He is survived by three children.

Dr. Eugene Albert Gilman, of Dorchester, died at his home June 17, aged 74 years. He was a graduate of Harvard Medical School in 1872 and joined the Massachusetts Medical Society in that year from South Boston. He was retired in 1907.

Dr. Frank D. Gray, president of the New Jersey Medical Society, died in Jersey City on June 11. He received the degree of M.D. from New York University in 1883.